Search Notes

Application No.	Applicant(s)	
09/460,216	ALLAWAY, G. P. ET AL.	
Examiner	**Art Unit**	
Jeffrey S. Parkin, Ph.D.	1648	

SEARCHED			
Class	Subclass	Date	Examiner
435	5	2/5/2005	
424	188.1		
	208.1		
UPDATED AS NECESSARY		01/22/06	J

INTERFERENCE SEARCHED			
Class	Subclass	Date	Examiner

SEARCH NOTES (INCLUDING SEARCH STRATEGY)		
	DATE	EXMR
USPATFUL WPIDS MEDLINE (updated as necessary)	2/5/2005	
UPDATED AS NECESSARY	01/22/06	J

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 NEWS  5  DEC 14  2006 MeSH terms loaded for MEDLINE file segment of TOXCENTER
 NEWS  6  DEC 14  CA/CAplus to be enhanced with updated IPC codes
 NEWS  7  DEC 21  IPC search and display fields enhanced in CA/CAplus with the
                  IPC reform
 NEWS  8  DEC 23  New IPC8 SEARCH, DISPLAY, and SELECT fields in USPATFULL/
                  USPAT2
 NEWS  9  JAN 13  IPC 8 searching in IFIPAT, IFIUDB, and IFICDB
 NEWS 10  JAN 13  New IPC 8 SEARCH, DISPLAY, and SELECT enhancements added to
                  INPADOC
 NEWS 11  JAN 17  Pre-1988 INPI data added to MARPAT
 NEWS 12  JAN 17  IPC 8 in the WPI family of databases including WPIFV

 NEWS EXPRESS  JANUARY 03 CURRENT VERSION FOR WINDOWS IS V8.01,
               CURRENT MACINTOSH VERSION IS V6.0c(ENG) AND V6.0Jc(JP),
               AND CURRENT DISCOVER FILE IS DATED 19 DECEMBER 2005.
               V8.0 USERS CAN OBTAIN THE UPGRADE TO V8.01 AT
               http://download.cas.org/express/v8.0-Discover/

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FILE 'HOME' ENTERED AT 13:34:37 ON 22 JAN 2006

=> file uspatful
COST IN U.S. DOLLARS                                SINCE FILE      TOTAL
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FILE 'USPATFULL' ENTERED AT 13:34:46 ON 22 JAN 2006
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FILE COVERS 1971 TO PATENT PUBLICATION DATE: 19 Jan 2006 (20060119/PD)
FILE LAST UPDATED: 19 Jan 2006 (20060119/ED)
HIGHEST GRANTED PATENT NUMBER: US6988280
HIGHEST APPLICATION PUBLICATION NUMBER: US2006015978
CA INDEXING IS CURRENT THROUGH 19 Jan 2006 (20060119/UPCA)
ISSUE CLASS FIELDS (/INCL) CURRENT THROUGH: 19 Jan 2006 (20060119/PD)
REVISED CLASS FIELDS (/NCL) LAST RELOADED: Dec 2005
USPTO MANUAL OF CLASSIFICATIONS THESAURUS ISSUE DATE: Dec 2005


=> e allaway g p/in
E1            1     ALLAWAY DAVID/IN
E2            1     ALLAWAY DAVID R/IN
E3            0 --> ALLAWAY G P/IN
E4           22     ALLAWAY GRAHAM P/IN
E5            1     ALLAWAY JAMES R/IN
E6            1     ALLAWAY JULIA B/IN
E7            4     ALLAWAY MICHAEL B/IN
E8            1     ALLAWAY MICHAEL J/IN
E9            1     ALLAWAY PHILIP N/IN
E10           1     ALLAWAY SCOTT/IN
E11           1     ALLAWAY STEVEN M/IN
E12           4     ALLAWI HATIM/IN

=> s e4
L1           22 "ALLAWAY GRAHAM P"/IN

=> d l1,cbib,1-22

L1   ANSWER 1 OF 22  USPATFULL on STN
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thereof and use thereof.
Allaway, Graham P., Darnestown, MD, UNITED STATES
Wild, Carl T., Gaithersburg, MD, UNITED STATES
Kashiwada, Yoshiki, Niigata, JAPAN
Lee, Kuo-Hsiung, Chapel Hill, NC, UNITED STATES
Panacos Pharmaceuticals, Inc., Gaithersburg, MD (U.S. corporation)The University of North Carolina at Chapel Hill, Chapel Hill, NC (U.S. corporation)Niigata University of Pharmacy and Applied Life Sciences, Niigata, JAPAN (U.S. corporation)
US 2005020548 A1 20050127
APPLICATION: US 2004-870555 A1 20040618 (10)
PRIORITY: US 2002-413451P 20020926 (60)
DOCUMENT TYPE: Utility; APPLICATION.
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L1 ANSWER 2 OF 22 USPATFULL on STN
2005:17637 Inhibition of HIV-1 replication by disruption of the processing of the viral capsid-spacer peptide 1 protein.
Salzwedel, Karl, Olney, MD, UNITED STATES
Li, Feng, Gaithersburg, MD, UNITED STATES
Wild, Carl T., Gaithersburg, MD, UNITED STATES
Allaway, Graham P., Darnestown, MD, UNITED STATES
Freed, Eric O., Frederick, MD, UNITED STATES
US 2005015039 A1 20050120
APPLICATION: US 2004-851637 A1 20040524 (10)
PRIORITY: US 2003-496660P 20030821 (60)
US 2003-443180P 20030129 (60)
DOCUMENT TYPE: Utility; APPLICATION.
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L1 ANSWER 3 OF 22 USPATFULL on STN
2004:334243 Inhibition of HIV-1 replication by disruption of the processing of the viral capsid-spacer peptide 1 protein.
Salzwedel, Karl, Olney, MD, UNITED STATES
Li, Feng, Gaithersburg, MD, UNITED STATES
Wild, Carl T., Gaithersburg, MD, UNITED STATES
Allaway, Graham P., Darnestown, MD, UNITED STATES
Freed, Eric O., Frederick, MD, UNITED STATES
US 2004265320 A1 20041230
APPLICATION: US 2004-766528 A1 20040129 (10)
PRIORITY: US 2003-496660P 20030821 (60)
US 2003-443180P 20030129 (60)
DOCUMENT TYPE: Utility; APPLICATION.
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L1 ANSWER 4 OF 22 USPATFULL on STN
2004:171850 Method for detecting viral inactivating agents.
Allaway, Graham P., Darnestown, MD, UNITED STATES
Wild, Carl T., Gaithersburg, MD, UNITED STATES
Salzwedel, Karl, Olney, MD, UNITED STATES
Panacos Pharmaceuticals, Inc. (U.S. corporation)
US 2004132011 A1 20040708
APPLICATION: US 2003-685801 A1 20031016 (10)
PRIORITY: US 2002-418341P 20021016 (60)
DOCUMENT TYPE: Utility; APPLICATION.
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L1 ANSWER 5 OF 22 USPATFULL on STN
2004:171469 Monoacylated betulin and dihydrobetulin derivatives, preparation thereof and use thereof.
Allaway, Graham P., Darnestown, MD, UNITED STATES
Wild, Carl T., Gaithersburg, MD, UNITED STATES
Kashiwada, Yoshiki, Niigata, JAPAN
Lee, Kuo-Hsiung, Chapel Hill, NC, UNITED STATES
Panacos Pharmaceuticals, Inc. (non-U.S. corporation)The University of North Carolina at Chapel Hill (non-U.S. corporation)Niigata University of Pharmacy and Applied Science (non-U.S. corporation)
US 2004131629 A1 20040708
APPLICATION: US 2003-670797 A1 20030926 (10)
PRIORITY: US 2002-413451P 20020926 (60)
DOCUMENT TYPE: Utility; APPLICATION.
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L1 ANSWER 6 OF 22 USPATFULL on STN
2004:113689 Uses of a chemokine receptor for inhibiting HIV-1 infection.
Allaway, Graham P., Mohegan Lake, NY, UNITED STATES
Dragic, Tatjana, Hartsdale, NY, UNITED STATES
Litwin, Virginia M., Fayetteville, NY, UNITED STATES
Maddon, Paul J., Elmsford, NY, UNITED STATES
Moore, John P., New York, NY, UNITED STATES
Trkola, Alexandra, New York, NY, UNITED STATES
Progenics Pharmaceuticals, Inc. (U.S. corporation)Aaron Diamond AIDS

US 2004086528 A1 20040506
APPLICATION: US 2001-852238 A1 20010509 (9)
PRIORITY: US 1996-19941P 19960614 (60)
DOCUMENT TYPE: Utility; APPLICATION.
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L1 ANSWER 7 OF 22 USPATFULL on STN
2003:64649 METHODS FOR USING RESONANCE ENERGY TRANSFER- BASED ASSAY OF HIV-1 ENVELOPE GYLCOPROTEIN-MEDIATED MEMBRANE FUSION, AND KITS FOR PRACTICING SAME.
ALLAWAY, GRAHAM P., MORETON MERSEYSIDE, UNITED KINGDOM
LITWIN, VIRGINIA M., FAYETTEVILLE, NY, UNITED STATES
MADDON, PAUL J., ELMSFORD, NY, UNITED STATES
US 2003044770 A1 20030306
APPLICATION: US 1999-412284 A1 19991005 (9)
DOCUMENT TYPE: Utility; APPLICATION.
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L1 ANSWER 8 OF 22 USPATFULL on STN
2003:11187 Substituted 3',4' -Di-O-camphanoyl-(+)-cis-khellactone analogs, compositions thereof, and methods for using thereof.
Lee, Kuo-Hsiung, Chapel Hill, NC, UNITED STATES
Xie, Lan, Beijing, CHINA
Allaway, Graham P., Darnestown, MD, UNITED STATES
Wild, Carl T., Gaithersburg, MD, UNITED STATES
Panacos Pharmaceuticals, Inc. (U.S. corporation)
US 2003008891 A1 20030109
APPLICATION: US 2002-96107 A1 20020313 (10)
PRIORITY: US 2001-275043P 20010313 (60)
DOCUMENT TYPE: Utility; APPLICATION.
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L1 ANSWER 9 OF 22 USPATFULL on STN
2002:279995 Method for preventing HIV-1 infection of CD4+ cells.
Allaway, Graham P., Mohegan Lake, NY, UNITED STATES
Litwin, Virginia M., Fayetteville, NY, UNITED STATES
Maddon, Paul J., Elmsford, NY, UNITED STATES
Olson, William C., Ossining, NY, UNITED STATES
Progenics Pharmaceuticals, Inc. (U.S. corporation)
US 2002155429 A1 20021024
APPLICATION: US 2001-888938 A1 20010625 (9)
PRIORITY: US 1996-19715P 19960614 (60)
US 1996-14532P 19960402 (60)
DOCUMENT TYPE: Utility; APPLICATION.
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L1 ANSWER 10 OF 22 USPATFULL on STN
2002:178733 Assay for detection of viral fusion inhibitors.
Wild, Carl T., Gaithersburg, MD, UNITED STATES
Allaway, Graham P., Darnestown, MD, UNITED STATES
US 2002094521 A1 20020718
APPLICATION: US 2001-779451 A1 20010209 (9)
PRIORITY: US 2000-235901P 20000928 (60)
US 2000-181543P 20000210 (60)
DOCUMENT TYPE: Utility; APPLICATION.
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L1 ANSWER 11 OF 22 USPATFULL on STN
2002:99580 Non-peptidyl moiety-conjugated CD4-gamma2 and CD4 -IgG2 immunoconjugates, and uses thereof.
Maddon, Paul J., New York, NY, UNITED STATES
Allaway, Graham P., Mohegan Lake, NY, UNITED STATES
Progenics Pharmacueticals, Inc. (U.S. corporation)
US 2002052481 A1 20020502
APPLICATION: US 2001-766995 A1 20010122 (9)
DOCUMENT TYPE: Utility; APPLICATION.
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L1 ANSWER 12 OF 22 USPATFULL on STN
2002:85121 Fluorescence resonance energy transfer screening assay for the identification of HIV-1 envelope glycoprotein-medicated cell.
Allaway, Graham P., Moreton Merseyside, UNITED KINGDOM
Litwin, Virginia M., Fayetteville, NY, UNITED STATES
Maddon, Paul J., Elmsford, NY, UNITED STATES
Progenics Pharmaceuticals, Inc. (non-U.S. corporation)
US 2002045161 A1 20020418
APPLICATION: US 2001-904356 A1 20010712 (9)
DOCUMENT TYPE: Utility; APPLICATION.
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L1 ANSWER 13 OF 22 USPATFULL on STN
2002:24365 Method for preventing HIV-1 infection of CD4+ cells.

Litwin, Virginia M., Fayetteville, NY, United States
Maddon, Paul J., Elmsford, NY, United States
Olson, William C., Ossining, NY, United States
Progenics Pharmaceuticals, Inc., Tarrytown, NY, United States (U.S. corporation)
US 6344545 B1 20020205
APPLICATION: US 1997-831823 19970402 (8)
PRIORITY: US 1996-19715P 19960614 (60)
US 1996-14532P 19960402 (60)
DOCUMENT TYPE: Utility; GRANTED.
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L1 ANSWER 14 OF 22 USPATFULL on STN
2002:19398 Non-peptidyl moiety-conjugated CD4-gamma2 and CD4-IgG2 immunoconjugates and uses thereof.
Allaway, Graham P., Mohegan Lake, NY, United States
Maddon, Paul J., New York, NY, United States
Progenics Pharmaceuticals, Inc., Westchester, NY, United States (U.S. corporation)
US 6342586 B1 20020129
APPLICATION: US 1999-409006 19990929 (9)
DOCUMENT TYPE: Utility; GRANTED.
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L1 ANSWER 15 OF 22 USPATFULL on STN
2002:17437 Method for generating immunogens that elicit neutralizing antibodies against fusion-active regions of HIV envelope proteins.
Wild, Carl T., Gaithersburg, MD, UNITED STATES
Allaway, Graham P., Darnestown, MD, UNITED STATES
US 2002010317 A1 20020124
APPLICATION: US 2001-809060 A1 20010316 (9)
PRIORITY: US 2000-189981P 20000317 (60)
DOCUMENT TYPE: Utility; APPLICATION.
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L1 ANSWER 16 OF 22 USPATFULL on STN
2001:218025 Compounds capable of inhibiting HIV-1 infection.
Litwin, Virginia M., Fayetteville, NY, United States
Allaway, Graham P., Cheshire, Great Britain
Maddon, Paul J., New York, NY, United States
Progenics Pharmaceuticals, Inc. (U.S. corporation)
US 2001046512 A1 20011129
APPLICATION: US 2001-891062 A1 20010625 (9)
DOCUMENT TYPE: Utility; APPLICATION.
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L1 ANSWER 17 OF 22 USPATFULL on STN
2001:112032 Fluorescence resonance energy transfer screening assay for the identification of compounds that are capable of abrogating macrophage-tropic HIV-1 cell fusion.
Allaway, Graham P., Moreton Merseyside, United Kingdom
Litwin, Virginia M., Fayetteville, NY, United States
Maddon, Paul J., Elmsford, NY, United States
Progenics Pharmaceuticals, Inc., Tarrytown, NY, United States (U.S. corporation)
US 6261763 B1 20010717
WO 9641020 19961219
APPLICATION: US 1998-973601 19980316 (8)
WO 1996-US9894 19960607 19980316 PCT 371 date 19980316 PCT 102(e) date
DOCUMENT TYPE: Utility; GRANTED.
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L1 ANSWER 18 OF 22 USPATFULL on STN
2001:11011 Non-peptidyl moiety-conjugated CD4-gamma2 and CD4-IgG2 immunoconjugates, and uses thereof.
Maddon, Paul J., New York, NY, United States
Allaway, Graham P., Mohegan Lake, NY, United States
Progenics Pharmaceuticals, Inc., Tarrytown, NY, United States (U.S. corporation)
US 6177549 B1 20010123
APPLICATION: US 1999-329916 19990610 (9)
DOCUMENT TYPE: Utility; Granted.
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L1 ANSWER 19 OF 22 USPATFULL on STN
2000:109525 Method for preventing HIV-1 infection of CD4+ cells.
Allaway, Graham P., Mohegan Lake, NY, United States
Litwin, Virginia M., Fayetteville, NY, United States
Maddon, Paul J., Elmsford, NY, United States
Olson, William C., Ossining, NY, United States
Progenics Pharmaceuticals, Inc., Tarrytown, NY, United States (U.S. corporation)

APPLICATION: US 1997-876078 19970613 (8)
PRIORITY: US 1996-19715P 19960614 (60)
US 1996-14532P 19960402 (60)
DOCUMENT TYPE: Utility; Granted.
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L1 ANSWER 20 OF 22 USPATFULL on STN
2000:83819 Non-peptidyl moiety-conjugated CD4-gamma2 and CD4-IgG2 Immunoconjugates, and uses thereof.
Allaway, Graham P., Mohegan Lake, NY, United States
Maddon, Paul J., New York, NY, United States
Progenics Pharmaceuticals, Inc., Tarrytown, NY, United States (U.S. corporation)
US 6083478 20000704
WO 9403191 19940217
APPLICATION: US 1996-379516 19960610 (8)
WO 1994-EP1349 19940429 19960610 PCT 371 date 19960610 PCT 102(e) date
DOCUMENT TYPE: Utility; Granted.
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L1 ANSWER 21 OF 22 USPATFULL on STN
2000:28118 Non-peptidyl moiety-conjugated CD4-gamma2 and CD4-IgG2 immunoconjugates, and uses thereof.
Maddon, Paul J., New York, NY, United States
Allaway, Graham P., Mohegan Lake, NY, United States
Progenics Pharmaceuticals, Inc., Tarrytown, NY, United States (U.S. corporation)
US 6034223 20000307
APPLICATION: US 1995-477460 19950607 (8)
DOCUMENT TYPE: Utility; Granted.
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L1 ANSWER 22 OF 22 USPATFULL on STN
1998:122515 Synergistic composition of CD4-based protein and anti-HIV-1 antibody, and methods of using same.
Allaway, Graham P., Mohegan Lake, NY, United States
Maddon, Paul J., New York, NY, United States
Progenics Pharmaceuticals, Inc., Tarrytown, NY, United States (U.S. corporation)
US 5817767 19981006
APPLICATION: US 1993-21879 19930224 (8)
DOCUMENT TYPE: Utility; Granted.
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

=> d l1,cbib,clm,9,13,16,19

L1 ANSWER 9 OF 22 USPATFULL on STN
2002:279995 Method for preventing HIV-1 infection of CD4+ cells.
Allaway, Graham P., Mohegan Lake, NY, UNITED STATES
Litwin, Virginia M., Fayetteville, NY, UNITED STATES
Maddon, Paul J., Elmsford, NY, UNITED STATES
Olson, William C., Ossining, NY, UNITED STATES
Progenics Pharmaceuticals, Inc. (U.S. corporation)
US 2002155429 A1 20021024
APPLICATION: US 2001-888938 A1 20010625 (9)
PRIORITY: US 1996-19715P 19960614 (60)
US 1996-14532P 19960402 (60)
DOCUMENT TYPE: Utility; APPLICATION.
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
CLM What is claimed is:
1. A method for inhibiting fusion of HIV-1 to CD4+ cells which comprises contacting CD4+ cells with a non-chemokine agent capable of binding to a chemokine receptor in an amount and under conditions such that fusion of HIV-1 to the CD4+ cells is inhibited.

2. A method for inhibiting HIV-1 infection of CD4+ cells which comprises contacting CD4+ cells with a non-chemokine agent capable of binding to a chemokine receptor in an amount and under conditions such that fusion of HIV-1 to the CD4+ cells is inhibited, thereby inhibiting HIV-1 infection.

3. The method of claim 1 or 2, wherein the non-chemokine agent is an oligopeptide.

4. The method of claim 1 or 2, wherein the non-chemokine agent is a polypeptide.

5. The method of claim 1 or 2, wherein the non-chemokine agent is an antibody or a portion of an antibody.

6. The method of claim 1 or 2, wherein the non-chemokine agent is a

7. A non-chemokine agent capable of binding to a chemokine receptor and inhibiting fusion of HIV-1 to CD4+ cells.

8. The non-chemokine agent of claim 7, wherein the non-chemokine agent is a oligopeptide.

9. The non-chemokine agent of claim 7, wherein the non-chemokine agent is a nonpeptidyl agent.

10. The non-chemokine agent of claim 7, wherein the non-chemokine agent is a polypeptide.

11. The non-chemokine agent of claim 10, wherein the polypeptide is an antibody or a portion of an antibody.

12. The non-chemokine agent of claim 10, wherein the polypeptide comprises amino acid sequence as set forth in SEQ ID NO:5.

13. The non-chemokine agent of claim 10, wherein the polypeptide comprises the MIP-1β sequence with the deletion of the first seven N-terminal amino acids of said sequence.

14. The non-chemokine agent of claim 10, wherein the polypeptide comprises the MIP-1β sequence with the deletion of the first eight N-terminal amino acids of said sequence.

15. The non-chemokine agent of claim 10, wherein the polypeptide comprises the MIP-1β sequence with the deletion of the first nine N-terminal amino acids of said sequence.

16. The non-chemokine agent of claim 10, wherein the polypeptide comprises the MIP-1β sequence with the deletion of the first ten N-terminal amino acids of said sequence.

17. The non-chemokine agent of claim 10, wherein the polypeptide comprises the MIP-1β sequence with the N-terminal sequence modified by addition of an amino acid or oligopeptide.

18. The non-chemokine agent of claim 10, wherein the polypeptide comprises the MIP-1β sequence with the N-terminal sequence modified by removing the N-terminal alanine and replacing it by serine or threonine and an additional amino acid or oligopeptide or nonpeptidyl moiety.

19. The non-chemokine agent of claim 17 or 18, wherein the additional amino acid is methionine.

20. An agent capable of binding to CXCR4 and inhibiting HIV-1 infection.

21. The agent of claim 20, wherein the agent is an oligopeptide.

22. The agent of claim 20, wherein the agent is a polypeptide.

23. The non-chemokine agent of claim 22, wherein the polypeptide comprises the SDF-1 sequence with the deletion of the first six N-terminal amino acids of said sequence.

24. The non-chemokine agent of claim 22, wherein the polypeptide comprises the SDF-1 sequence with the deletion of the first seven N-terminal amino acids of said sequence.

25. The non-chemokine agent of claim 22, wherein the polypeptide comprises the SDF-1 sequence with the deletion of the first eight N-terminal amino acids of said sequence.

26. The non-chemokine agent of claim 22, wherein the polypeptide comprises the SDF-1 sequence with the deletion of the first nine N-terminal amino acids of said sequence.

27. The non-chemokine agent of claim 22, wherein the N-terminal glycine of SDF-1 is replaced by serine and derivatized with biotin.

28. The non-chemokine agent of claim 22, wherein the N-terminal glycine of SDF-1 is replaced by serine and derivatized with methionine.

29. The non-chemokine agent of claim 22, wherein the N-terminus of SDF-1 is modified by the addition of a methionine before the terminal glycine.

30. The agent of claim 22, wherein the agent is an antibody or a portion of an antibody.

31. The agent of claim 20, wherein the agent is a non-peptidyl agent.

32. A pharmaceutical composition comprising an amount of the non-chemokine agent of claim 7 effective to inhibit fusion of HIV-1 to CD4+ cells and a pharmaceutically acceptable carrier.

33. A pharmaceutical composition comprising an amount of the non-chemokine agent of claim 20 effective to inhibit fusion of HIV-1 to CD4+ cells and a pharmaceutically acceptable carrier.

34. A composition of matter capable of binding to a chemokine receptor and inhibiting fusion of HIV-1 to CD4+ cells comprising a non-chemokine agent linked to a ligand capable of binding to a cell surface receptor of the CD4+ cells other than the chemokine receptor such that the binding of the non-chemokine agent to the chemokine receptor does not inhibit the binding of the ligand to the other receptor.

35. The composition of matter of claim 34, wherein the cell surface receptor is CD4.

36. The composition of matter of claim 34, wherein the ligand comprises an antibody or a portion of an antibody.

37. A pharmaceutical composition comprising an amount of the composition of matter of claim 34 effective to inhibit fusion of HIV-1 to CD4+ cells and a pharmaceutically acceptable carrier.

38. A composition of matter capable of binding to the chemokine receptor and inhibiting fusion of HIV-1 to CD4+ cells comprising a non-chemokine agent linked to a compound capable of increasing the in vivo half-life of the non-chemokine agent.

39. The composition of matter of claim 38, wherein the compound is polyethylene glycol.

40. A pharmaceutical composition comprising an amount of the composition of claim 38 effective to inhibit fusion of HIV-1 to CD4+ cells and a pharmaceutically acceptable carrier.

41. A method for reducing the likelihood of HIV-1 infection in a subject comprising administering the pharmaceutical composition of claim 32, 33, 37 or 40 to the subject.

42. A method for treating HIV-1 infection in a subject comprising administering the pharmaceutical composition of claim 32, 33, 39 or 40 to the subject.

43. A method for determining whether a non-chemokine agent is capable of inhibiting the fusion of HIV-1 to a CD4+ cell which comprises: (a) contacting (i) a CD4+ cell, which is labeled with a first dye, with (ii) a cell expressing the HIV-1 envelope glycoprotein on its surface, which is labeled with a second dye, in the presence of an excess of the agent under conditions permitting the fusion of the CD4+ cell to the cell expressing the HIV-1 envelope glycoprotein on its surface in the absence of the agent, the first and second dyes being selected so as to allow resonance energy transfer between the dyes; (b) exposing the product of step (a) to conditions which would result in resonance energy transfer if fusion has occurred; and (c) determining whether there is a reduction of resonance energy transfer, when compared with the resonance energy transfer in the absence of the agent, a decrease in transfer indicating that the agent is capable of inhibiting fusion of HIV-1 to CD4+ cells.

44. The method of claim 43, wherein the agent is an oligopeptide.

45. The method of claim 43, wherein the agent is a polypeptide.

46. The method of claim 43, wherein the agent is an antibody or a portion of an antibody.

47. The method of claim 43, wherein the agent is a nonpeptidyl agent.

48. The method of claim 43, wherein the CD4+ cell is a PM1 cell.

49. The method of claim 43, wherein the cell expressing the HIV-1 envelope glycoprotein is a HeLa cell expressing $HIV\text{-}1_{JR\text{-}FL}$ gp120/gp41.

50. The method of claim 43, wherein the cell expressing the HIV-1 envelope glycoprotein is a HeLa cell expressing $HIV\text{-}1_{LAI}$ gp120/gp41.

L1 ANSWER 13 OF 22 USPATFULL on STN
2002:24365 Method for preventing HIV-1 infection of CD4+ cells.
Allaway, Graham P., Mohegan Lake, NY, United States
Litwin, Virginia M., Fayetteville, NY, United States
Maddon, Paul J., Elmsford, NY, United States
Olson, William C., Ossining, NY, United States
Progenics Pharmaceuticals, Inc., Tarrytown, NY, United States (U.S. corporation)
US 6344545 B1 20020205
APPLICATION: US 1997-831823 19970402 (8)
PRIORITY: US 1996-19715P 19960614 (60)
US 1996-14532P 19960402 (60)
DOCUMENT TYPE: Utility; GRANTED.
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
CLM What is claimed is:
1. A method of inhibiting HIV-1 infection of a CD4+ cell which comprises contacting the CD4+ cell with an antibody or portion of an antibody capable of binding to a chemokine receptor on the surface of the CD4+ cell in an amount and under conditions such that fusion of HIV-1 or an HIV-1 infected cell to the CD4+ cell is inhibited, thereby inhibiting HIV-1 infection of the CD4+ cell.

2. The method of claim 1, wherein the chemokine receptor is a CCR5 chemokine receptor.

3. The method of claim 1, wherein the CD4+ cell is a PM-1 cell.

4. The method of claim 1, wherein the CD4+ cell is a primary CD4+ T-cell.

5. The method of claim 1, wherein the CD4+ cell is a PMBC cell.

6. The method of claim 1, wherein the antibody is a monoclonal antibody.

L1 ANSWER 16 OF 22 USPATFULL on STN
2001:218025 Compounds capable of inhibiting HIV-1 infection.
Litwin, Virginia M., Fayetteville, NY, United States
Allaway, Graham P., Cheshire, Great Britain
Maddon, Paul J., New York, NY, United States
Progenics Pharmaceuticals, Inc. (U.S. corporation)
US 2001046512 A1 20011129
APPLICATION: US 2001-891062 A1 20010625 (9)
DOCUMENT TYPE: Utility; APPLICATION.
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
CLM What is claimed is:
1. An antibody capable of specifically inhibiting the fusion of an HIV-1 envelope glycoprotein cell with an appropriate CD4+ cell without cross reacting with the HIV-1 envelope glycoprotein or CD4 and capable of inhibiting infection by one or more strains of HIV-1.

2. A monoclonal antibody of claim i.

3. A hybridoma cell line producing the monoclonal antibody of claim 2.

4. A chimeric monoclonal antibody of claim 2.

5. A humanized monoclonal antibody of claim 4.

6. A human monoclonal antibody of claim 2.

7. A single chain antibody or an antigen binding antibody fragment of claim 2.

8. A monoclonal antibody capable of competitively inhibiting the binding of the monoclonal antibody of claim 2 to its target molecule.

9. The monoclonal antibody of claim 2, 4, 3, 6, 7, or 8 labelled with a detectable marker.

10. A monoclonal antibody of claim 9 wherein the detectable marker is a radioactive isotope, enzyme, dye or biotin.

11. A pharmaceutical composition comprising the complete or a portion of the monoclonal antibody of claim 2, 4, 5, 6, 7 or 8 and a pharmaceutically acceptable carrier.

12. A method of inhibiting HIV-1 infection in a subject comprising administering an effective amount of the pharmaceutical composition of claim 11 to the subject.

of the light chain protein of the monoclonal antibody of claim 2, 4, 5, 6 or 8.

14. An isolated nucleic acid molecule encoding the complete of a portion of the heavy chain protein of the monoclonal antibody of claim 2, 4, 5, 6 or 8.

15. An isolated nucleic acid molecule encoding the single chain antibody of claim 7.

16. A vector comprising the nucleic acid molecule of claim 13, 14 or 15 operably linked to a promoter of RNA transcription.

17. A vector comprising the nucleic acid molecules of claims 13 and 14 each operably linked to a promoter of RNA transcription.

18. A host vector system comprising one or more vectors of claim 16 or 17 in a suitable host cell.

19. A host vector system of claim 18, wherein the suitable host cell is selected from a group consisting of a bacterial cell, an insect cell, a yeast cell or a mammalian cell.

20. The molecule specifically recognized by nhe monoclonal antibody of claim 1.

21. A glycoloipd molecule of claim 20.

22. A polypeptide molecule of claim 20.

23. An isolated nucleic acid molecule encoding the complete or a portion of the polypeptide of claim 22.

24. A multichain polypeptide molecule comprising the polypeptide of claim 22.

25. A soluble protein comprising a portion of the polypep-ide of claim 22 or 24.

26. A pharmaceutical composition comprising an effective amount of the soluble protein of claim 25 to inhibit HIV-1 infection and a pharmaceutically acceptable carrier.

27. A method of inhibiting HIV-1 infection in a subject comprising administering an effective amount of the pharmaceutical composition of claim 26 to the subject.

28. An isolated nucleic acid molecule encoding the complete or a portion of a polypeptide of the multichain polypeptide molecule of claim 24.

29. A vector comprising the nucleic acid molecule of claim 23 or 28 operably linked to a promoter of RNA transcription.

30. A host vector system comprising the vector of claim 29 in a suitable host cell.

31. A host vector system of claim 30, wherein the suitable host cell is a selected from a group consisting of a bacterial cell, an insect cell, a yeast cell or a mammalian cell.

32. A method for identifying inhibitors of HIV-1 infection comprising steps of: (a) contacting an effective amount of a compound with a system which contains HIV-1 gp120, HIV-1 gp41 or a fragment thereof with the molecule of claim 20 under conditions permitting formation of a complex between HIV-1 gp120, HIV-1 gp41 or a fragment thereof and the molecule, so as to inhibit such formation; and (b) determining the amount of complex formed; and (c) comparing the amount determined in step (b) with the control which is without the addition of the compound, a decrease in the complex formation indicating that the compound is capable of inhibiting HIV-1 infection.

33. A method of claim 32, wherein the compound is not previously known.

34. The compound identified by claim 33.

35. A pharmaceutical composition comprising the compound identified by the method of claim 32 and a pharmaceutically acceptable carrier.

36. A method of inhibiting HIV-1 infection in a subject comprising administering an effective amount of the pharmaceutical composition of claim 35 to the subject.

in separate compartments: (a) purified HIV-1 gp120, HIV-1 gp41 or a fragment thereof; and (b) the molecule of claim 20.

38. A transgenio nonhuman animal which comprises an isolated DNA molecule encoding the molecule of claim 22 or 24.

39. The transgenic nonhuman animal of claim 38 further comprising an isolated DNA molecule encoding the full-length or portion of the CD4 molecule sufficient for binding the HIV-1 envelope glycoprotein.

L1 ANSWER 19 OF 22 USPATFULL on STN
2000:109525 Method for preventing HIV-1 infection of CD4+ cells.
Allaway, Graham P., Mohegan Lake, NY, United States
Litwin, Virginia M., Fayetteville, NY, United States
Maddon, Paul J., Elmsford, NY, United States
Olson, William C., Ossining, NY, United States
Progenics Pharmaceuticals, Inc., Tarrytown, NY, United States (U.S. corporation)
US 6107019 20000822
APPLICATION: US 1997-876078 19970613 (8)
PRIORITY: US 1996-19715P 19960614 (60)
US 1996-14532P 19960402 (60)
DOCUMENT TYPE: Utility; Granted.
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
CLM What is claimed is:
1. An in vitro method for determining whether an agent is capable of inhibiting HIV-1 infection of a CD4+ cell susceptible to HIV-1 infection comprising the steps of: (a) fixing a chemokine receptor on a solid matrix wherein the chemokine receptor is a co-receptor for HIV-1 infection; (b) contacting the fixed chemokine receptor with the agent under conditions permitting binding of the agent to the chemokine receptor; (c) removing any unbound agent; (d) contacting the resulting fixed chemokine receptor to which the agent is bound with a predetermined amount of gp120/CD4+ complex under conditions permitting binding of gp120/CD4 + complex to the fixed chemokine receptor in the absence of the agent; (e) removing any unbound gp120/CD4+ complex; (f) measuring the amount of gp120/CD4+ complex bound to the fixed chemokine receptor; and (g) comparing the amount measured in step (f) with the amount measured in the absence of the agent, a decrease in the amount bound in the presence of the agent indicating that the agent is capable of inhibiting HIV-1 infection.

2. An in vitro method for determining whether an agent is capable of inhibuting HIV-1 infection of a CD4+ cell susceptible to HIV-1 infection comprising the steps: (a) fixing a chernokine receptor on a solid matrix wherein the chemokine receptor is a co-receptor for HIV-1 infection; (b) contacting the fixed chemokine receptor with the agent and a predetermined amount of gp120/CD4+ complex under conditions permitting binding of the gp120/CD4+ complex to the fixed chemokine receptor in the absence of the agent; (c) removing any unbound agent or unbound gp120/CD4+ complex or both; (d) measuring the amount of gp120/CD4+ complex bound to the fixed chemokine receptor; and (e) comparing the amount measured in step (d) with the amount measured in the absence of the agent, a decrease in the amount bound in the presence of the agent indicating that the agent is capable of inhibiting HIV-1 infection.

3. An in vitro method for determining whether an agent is capable of inhibiting HIV-1 infection of a CD4+ cell susceptible to HIV-1 infection comprising steps of: (a) fixing a gp120/CD4+ complex on a solid matrix; (b) contacting the fixed gp120/CD4+ complex with the agent under conditions permitting the binding of the agent to the gp120/CD4+ complex; (c) removing any unbound agent; (d) contacting the resulting fixed gp120/CD4+ complex to which the agent is bcund with a predetermined amount of chemokine receptor, wherein the chemokine receptor is a co-receptor for HIV-1 infection, under conditions permitting binding of the chemokine receptor to the fixed the gp120/CD4+ complex in the absence of the agent; (e) removing any unbound chemokine receptor; (f) measuring the amount of chemokine receptor bound to the fixed gp120/CD4+ ; and (g) comparing the amount measured in step (f) with the amount measured in the absence of the agent, a decrease in the amount bound in the presence of the agent indicating that the agent is capable of inhibiting HIV-1 infection.

4. An in vitro method for determining whether an agent is capable of inhibiting HIV-1 infection of a CD4+ cell susceptible to HIV-1 infection comprising steps of: (a) fixing a gp120/CD4+ complex on a solid matrix: (b) contacting the fixed gp120/CD4+ complex with the agent and a predetermined amount of chemokine receptor, wherein the chemokine receptor is a co-receptor for HIV-1 infection, under co)nditions permitting binding of the chemokine receptor to the fixed

unbound agent or any unbound chemokine receptor or both: (d) measuring the amount of chemorkine receptor bound to the fixed gp120/CD4+ ; and (e) comparing the amount measured in step (d) with the amount measured in the absence of the agent, a decrease in the amount bound in the presence of the agent indicating that the agent is capable of inhibiting HIV-1 infection.

5. The method of claim 1, 2, 3, or 4 wherein the CD4+ is a soluble CD4+.

6. The method of claim 1, 2, 3, or 4 wherein the chemokine receptor is expressed on a cell.

7. The method of claim 6 wherein the cell is a L1.2 cell.

8. The method of claim 1 or 2, wherein the gp120, CD4+ or both are labeled with a detectable marker.

9. The method of claim 3 or 4 wherein the chemokine receptor is labeled with a detectable marker.

10. The method of claim 1 or 2, wherein the gp120, CD4+ or both are labeled with biotin.

11. The method of claim 2 or 4 wherein the chemokine receptor is labeled with biotin.

12. The method of any one of claims 1, 2, 3, or 4, wherein the chemockine receptor is CCR5.

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=> e litwin v m/in
E1            1     LITWIN STANLEY M/IN
E2            1     LITWIN STASZEWSKA ELZBIETA/IN
E3            0 --> LITWIN V M/IN
E4            9     LITWIN VIRGINIA M/IN
E5            2     LITWIN WALTER J/IN
E6            1     LITWIN WILLIAM J/IN
E7            1     LITWIN WILLIAM S/IN
E8            3     LITWIN WITOLD/IN
E9            3     LITWIN YORAM/IN
E10           1     LITWINCHUK ALEXANDER/IN
E11           1     LITWINETZ DENNIS M/IN
E12           1     LITWINOWICH MICHAEL J/IN

=> s e4
L2            9 "LITWIN VIRGINIA M"/IN

=> s l2 not l1
L3            1 L2 NOT L1

=> d l3,cbib

L3   ANSWER 1 OF 1  USPATFULL on STN
1998:72420 Antibodies to mammalian NK antigens and uses.
    Litwin, Virginia M., Palo Alto, CA, United States
    Gumperz, Jennifer E., Oakland, CA, United States
    Parham, Peter R., Stanford, CA, United States
    Phillips, Jr., Joseph H., San Carlos, CA, United States
    Lanier, Lewis L., Los Altos, CA, United States
    Schering Corporation, Kenilworth, NJ, United States (U.S. corporation)The
    Board of Trustees of The Leland Stanford Junior University, Palo Alto, CA,
    United States (U.S. corporation)
    US 5770387 19980623
    APPLICATION: US 1996-670987  19960628 (8)
    DOCUMENT TYPE: Utility; Granted.
CAS INDEXING IS AVAILABLE FOR THIS PATENT.


=> e maddon p j/in
E1            6     MADDOCKS THOMAS C/IN
E2            1     MADDON CHESTER L/IN
E3            0 --> MADDON P J/IN
E4           47     MADDON PAUL J/IN
E5            1     MADDOUX DON/IN
E6            1     MADDOUX LILLA A/IN
E7            3     MADDOX A DALE/IN
E8            1     MADDOX AL/IN
E9            1     MADDOX ALBERT F/IN
E10           1     MADDOX BRAIN L/IN
E11           1     MADDOX BRIAN L/IN
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=> s e4
L4          47 "MADDON PAUL J"/IN

=> s l4 not (l1 or l2)
L5          33 L4 NOT (L1 OR L2)

=> s l5 and (CCR5 or CC-CKR-5 or CKR5)
         1930 CCR5
       164045 CC
          589 CKR
      4208072 5
          148 CC-CKR-5
                (CC(W)CKR(W)5)
          103 CKR5
L6          11 L5 AND (CCR5 OR CC-CKR-5 OR CKR5)

=> d l6,cbib,1-11

L6   ANSWER 1 OF 11  USPATFULL on STN
2005:104593 Human immunodeficiency virus envelope clycoprotein mutants and uses
    thereof.
    Moore, John P., New York, NY, UNITED STATES
    Binley, James M., San Diego, CA, UNITED STATES
    Lu, Min, New York, NY, UNITED STATES
    Olson, William C., New York, NY, UNITED STATES
    Schulke, Norbert, New City, NY, UNITED STATES
    Gardner, Jason, Ardsley, NY, UNITED STATES
    Maddon, Paul J., Scarsdale, NY, UNITED STATES
    Sanders, Rogier, Amsterdam, NETHERLANDS
    US 2005089526 A1 20050428
    APPLICATION: US 2003-489040  A1  20020906 (10)
    WO 2002-US28331 20020906
    PRIORITY: US 2003-317909P  20010906 (60)
    US 2003-317764P  20010906 (60)
    US 2003-317910P  20010906 (60)
    US 2003-317775P  20010906 (60)
    US 2003-370264P  20020405 (60)
    US 2003-370410P  20020405 (60)
    DOCUMENT TYPE: Utility; APPLICATION.
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L6   ANSWER 2 OF 11  USPATFULL on STN
2004:291789 Synergistic inhibition of HIV-1 fusion and attachment, compositions
    and antibodies thereto.
    Olson, William C., Ossining, NY, UNITED STATES
    Maddon, Paul J., Scarsdale, NY, UNITED STATES
    Progenics Pharmaceuticals, Inc. (U.S. corporation)
    US 2004228869 A1 20041118
    APPLICATION: US 2004-763545  A1  20040123 (10)
    PRIORITY: US 1998-112532P  19981216 (60)
    DOCUMENT TYPE: Utility; APPLICATION.
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L6   ANSWER 3 OF 11  USPATFULL on STN
2004:286146 Stabilized viral envelope proteins and uses thereof.
    Binley, James M., Brooklyn, NY, UNITED STATES
    Schuelke, Norbert, New City, NY, UNITED STATES
    Olson, William C., Ossining, NY, UNITED STATES
    Maddon, Paul J., Scarsdale, NY, UNITED STATES
    Moore, John P., New York, NY, UNITED STATES
    Progenics Pharmaceuticals, Inc. (U.S. corporation)Aaron Diamond AIDS
    Research Centre (U.S. corporation)
    US 2004224308 A1 20041111
    APPLICATION: US 2004-780993  A1  20040218 (10)
    PRIORITY: US 1999-141168P  19990625 (60)
    DOCUMENT TYPE: Utility; APPLICATION.

L6   ANSWER 4 OF 11  USPATFULL on STN
2004:82318 Compositions and methods for inhibition of HIV-1 infection.
    Olson, William C., Ossining, NY, UNITED STATES
    Maddon, Paul J., Scarsdale, NY, UNITED STATES
    Progenics Pharmaceuticals, Inc. (U.S. corporation)
    US 2004062767 A1 20040401
    APPLICATION: US 2003-681879  A1  20031009 (10)
    DOCUMENT TYPE: Utility; APPLICATION.
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L6   ANSWER 5 OF 11  USPATFULL on STN
2004:72662 Stabilized viral envelope proteins and uses thereof.
    Binley, James M., Brooklyn, NY, United States
    Schuelke, Norbert, New City, NY, United States
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Maddon, Paul J., Scarsdale, NY, United States
Moore, John P., New York, NY, United States
Progenics Pharmaceuticals, Inc., Tarrytown, NY, United States (U.S. corporation)Aaron Diamond AIDS Research Centre (ADARC), New York, NY, United States (U.S. corporation)
US 6710173 B1 20040323
APPLICATION: US 2000-602864 20000623 (9)
PRIORITY: US 1999-141168P 19990625 (60)
DOCUMENT TYPE: Utility; GRANTED.

L6 ANSWER 6 OF 11 USPATFULL on STN
2003:324332 Anti-**CCR5** antibody.
Olson, William C., Ossining, NY, UNITED STATES
Maddon, Paul J., Scarsdale, NY, UNITED STATES
Tsurushita, Naoya, Palo Alto, CA, UNITED STATES
Hinton, Paul R., Sunnyvale, CA, UNITED STATES
Vasquez, Maximiliano, Palo Alto, CA, UNITED STATES
US 2003228306 A1 20031211
APPLICATION: US 2003-371483 A1 20030221 (10)
DOCUMENT TYPE: Utility; APPLICATION.
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L6 ANSWER 7 OF 11 USPATFULL on STN
2003:119700 Compositions and methods for inhibition of hiv-1 infection.
Olson, William C., Ossining, NY, UNITED STATES
Maddon, Paul J., Scarsdale, NY, UNITED STATES
US 2003082185 A1 20030501
APPLICATION: US 2000-493346 A1 20000128 (9)
DOCUMENT TYPE: Utility; APPLICATION.
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L6 ANSWER 8 OF 11 USPATFULL on STN
2003:76924 Stabilized viral envelope proteins and uses thereof.
Binley, James M., Brooklyn, NY, UNITED STATES
Schuelke, Norbert, New City, NY, UNITED STATES
Olson, William C., Ossining, NY, UNITED STATES
Maddon, Paul J., Scarsdale, NY, UNITED STATES
Moore, John P., New York, NY, UNITED STATES
Progenics Pharmaceuticals, Inc., Aaron Diamonds AIDS Research Center (ADARC) (U.S. corporation)
US 2003052839 A1 20030320
APPLICATION: US 2001-32162 A1 20011221 (10)
DOCUMENT TYPE: Utility; APPLICATION.

L6 ANSWER 9 OF 11 USPATFULL on STN
2003:64291 Methods for inhibiting HIV-1 infection.
Olson, William C., Ossining, NY, UNITED STATES
Maddon, Paul J., Scarsdale, NY, UNITED STATES
US 2003044411 A1 20030306
APPLICATION: US 2002-116797 A1 20020405 (10)
PRIORITY: US 2001-282380P 20010406 (60)
DOCUMENT TYPE: Utility; APPLICATION.
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L6 ANSWER 10 OF 11 USPATFULL on STN
2002:265543 Methods for inhibiting HIV-1 infection.
Olson, William C., Ossining, NY, UNITED STATES
Maddon, Paul J., Scarsdale, NY, UNITED STATES
US 2002146415 A1 20021010
APPLICATION: US 2001-828615 A1 20010406 (9)
DOCUMENT TYPE: Utility; APPLICATION.
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L6 ANSWER 11 OF 11 USPATFULL on STN
2002:198280 Compositions and methods for inhibition of HIV-1 infection.
Olson, William C., Ossining, NY, UNITED STATES
Maddon, Paul J., Scarsdale, NY, UNITED STATES
US 2002106374 A1 20020808
APPLICATION: US 2001-912824 A1 20010725 (9)
PRIORITY: US 2001-266738P 20010206 (60)
DOCUMENT TYPE: Utility; APPLICATION.
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

=> d l6,cbib,clm,4,6,7,9-11

L6 ANSWER 4 OF 11 USPATFULL on STN
2004:82318 Compositions and methods for inhibition of HIV-1 infection.
Olson, William C., Ossining, NY, UNITED STATES
Maddon, Paul J., Scarsdale, NY, UNITED STATES
Progenics Pharmaceuticals, Inc. (U.S. corporation)
US 2004062767 A1 20040401

DOCUMENT TYPE: Utility; APPLICATION.
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
CLM What is claimed is:

1. A composition which comprises an admixture of two compounds, wherein one compound retards attachment of HIV-1 to a CD4+ cell by retarding binding of HIV-1 gp120 envelope glycoprotein to CD4 on the surface of the CD4+ cell and the other compound retards gp41 from adopting a conformation capable of mediating fusion of HIV-1 to a CD4+ cell by binding noncovalently to an epitope on a gp41 fusion intermediate, wherein the relative mass ratio of the compounds in the admixture ranges from about 100:1 to about 1:100, the composition being effective to inhibit HIV-1 infection of the CD4+ cell.

2. The composition of claim 1, wherein the compound which retards attachment of HIV-1 to the CD4+ cell by retarding binding of HIV-1 gp120 envelope glycoprotein to CD4 on the surface of the CD4+ cell is a CD4-based protein.

3. The composition of claim 2, wherein the CD4-based protein is a CD4-immunoglobulin fusion protein.

4. The composition of claim 3, wherein the CD4-immunoglobulin fusion protein is CD4-IgG2, wherein the CD4-IgG2 comprises two heavy chains and two lights chains, wherein the heavy chains are encoded by an expression vector designated CD4-IgG2HC-pRcCMV (ATCC Accession No. 75193) and the light chains are encoded by an expression vector designated CD4-kLC-pRcCMV (ATCC Accession No. 75194).

5. The composition of claim 1, wherein the compound which retards attachment of HIV-1 to the CD4+ cell by retarding binding of HIV-1 gp120 envelope glycoprotein to CD4 on the surface of the CD4+ cell is a protein, the amino acid sequence of which comprises that of a protein found in HIV-1 as an-envelope glycoprotein.

6. The composition of claim 5, wherein the protein binds to an epitope of CD4 on the surface of the CD4+ cell.

7. The composition of claim 6, wherein the envelope glycoprotein is selected from the group consisting of gp120, gp160, and gp140.

8. The composition of claim 1, wherein the compound which retards the attachment of HIV-1 to the CD4+ cell by retarding binding of HIV-1 gp120 envelope glycoprotein to CD4 on the surface of the CD4+ cell is an antibody or portion of an antibody.

9. The composition of claim 8, wherein the antibody is a monoclonal antibody.

10. The composition of claim 9, wherein the monoclonal antibody is a human, humanized or chimeric antibody.

11. The composition of claim 8, wherein the portion of the antibody is a Fab fragment of the antibody.

12. The composition of claim 8, wherein the portion of the antibody comprises the variable domain of the antibody.

13. The composition of claim 8, wherein the portion of the antibody comprises a CDR portion of the antibody.

14. The composition of claim 9, wherein the monoclonal antibody is an IgG, IgM, IgD, IgA, or IgE monoclonal antibody.

15. The composition of claim 9, wherein the monoclonal antibody binds to an HIV-1 envelope glycoprotein.

16. The composition of claim 15, wherein the HIV-1 envelope glycoprotein is selected from the group consisting of gp120 and gp160.

17. The composition of claim 16, wherein HIV-1 envelope glycoprotein is gp120 and the monoclonal antibody which binds to gp120 is IgG1b12 or F105.

18. The composition of claim 8, wherein the antibody binds to an epitope of CD4 on the surface of the CD4+ cell.

19. The composition of claim 1, wherein the compound which retards attachment of HIV-1 to the CD4+ cell by retarding binding of HIV-1 gp120 envelope glycoprotein to CD4 on the surface of the CD4+ cell is a peptide.

20. The composition of claim 1, wherein the compound which retards

envelope glycoprotein to CD4 on the surface of the CD4+ cell is a nonpeptidyl agent.

21. The composition of claim 1, wherein the compound which retards gp41 from adopting a conformation capable of mediating fusion of HIV-1 to a CD4+ cell by binding noncovalently to an epitope on a gp41 fusion intermediate is an antibody.

22. The composition of claim 21, wherein the antibody is a monoclonal antibody.

23. The composition of claim 1, wherein the compound which retards gp41 from adopting a conformation capable of mediating fusion of HIV-1 to a CD4+ cell by binding noncovalently to an epitope on a gp41 fusion intermediate is a peptide.

24. The composition of claim 1, wherein the compound which retards gp41 from adopting a conformation capable of mediating fusion of HIV-1 to a CD4+ cell by binding noncovalently to an epitope on a gp41 fusion intermediate is a fusion protein which comprises a peptide selected from the group consisting of T-20 (SEQ ID NO: 1), DP107 (SEQ ID NO: 2), N34 (SEQ ID NO: 3), C28 (SEQ ID NO: 4), and N34(L6)C28 (SEQ ID NO: 5).

25. The composition of claim 23, wherein the peptide is selected from the group consisting of T-20 (SEQ ID NO: 1), DP107 (SEQ ID NO: 2), N34 (SEQ ID NO: 3), C28 (SEQ ID NO: 4), and N34(L6)C28 (SEQ ID NO: 5).

26. The composition of claim 23, wherein the peptide is T-20 (SEQ ID NO: 1).

27. The composition of claim 1, wherein the compound which retards gp41 from adopting a conformation capable of mediating fusion of HIV-1 to a CD4+ cell by binding noncovalently to an epitope on a gp41 fusion intermediate is a non-peptidyl agent.

28. The composition of claim 1, wherein the relative mass ratio of each such compound in the admixture ranges from about 25:1 to about 1:1.

29. The composition of claim 28, wherein the mass ratio is about 25:1.

30. The composition of claim 28, wherein the mass ratio is about 5:1.

31. The composition of claim 28, wherein the mass ratio is about 1:1.

32. The composition of claim 1, wherein the composition is admixed with a carrier.

33. The composition of claim 32, wherein the carrier is an aerosol, intravenous, oral or topical carrier.

34. A method of inhibiting HIV-1 infection of a CD4+ cell which comprises contacting the CD4+ cell with an amount of the composition of claim 1 effective to inhibit HIV-1 infection of the CD4+ cell so as to thereby inhibit HIV-1 infection of the CD4+ cell.

35. The method of claim 34, wherein the CD4+ cell is present in a subject and the contacting is effected by administering the composition to the subject.

36. The method of claim 33, wherein the effective amount of the composition comprises from about 0.000001 mg/kg body weight to about 100 mg/kg body weight of the subject.

37. A method of inhibiting HIV-1 infection of a CD4+ cell which comprises contacting the CD4+ cell with an amount of a compound which retards attachment of HIV-1 to the CD4+ cell by retarding binding of HIV-1 gp120 envelope glycoprotein to CD4 on the surface of the CD4+ cell effective to inhibit HIV-1 infection of the CD4+ cell and an amount of a compound which retards gp41 from adopting a conformation capable of mediating fusion of HIV-1 to a CD4+ cell by binding noncovalently to an epitope on a gp41 fusion intermediate so as to thereby inhibit HIV-1 infection of the CD4+ cell.

38. The method of claim 37, wherein the CD4+ cell is present in a subject and the contacting is effected by administering the compounds to the subject.

39. The method of claim 38, wherein the compounds are administered to the subject simultaneously.

40. The method of claim 38, wherein the compounds are administered to the subject at different times.

41. The method of claim 38, wherein the compounds are administered to the subject by different routes of administration.

L6 ANSWER 6 OF 11 USPATFULL on STN
2003:324332 Anti-**CCR5** antibody.
Olson, William C., Ossining, NY, UNITED STATES
Maddon, Paul J., Scarsdale, NY, UNITED STATES
Tsurushita, Naoya, Palo Alto, CA, UNITED STATES
Hinton, Paul R., Sunnyvale, CA, UNITED STATES
Vasquez, Maximiliano, Palo Alto, CA, UNITED STATES
US 2003228306 A1 20031211
APPLICATION: US 2003-371483 A1 20030221 (10)
DOCUMENT TYPE: Utility; APPLICATION.
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
CLM What is claimed is:

1. An anti-**CCR5** antibody which comprises (i) two light chains, each light chain comprising the expression product of a plasmid designated pVK:HuPRO140-VK (ATCC Deposit Designation PTA-4097), and (ii) two heavy chains, each heavy chain comprising the expression product of either a plasmid designated pVg1:HuPRO140 HG2-VH (ATCC Deposit Designation PTA-4098) or a plasmid designated pVg1:HuPRO140 (mut B+D+I)-VH (ATCC Deposit Designation PTA-4099), or a fragment of such antibody, which binds to **CCR5** on the surface of a human cell.

2. The anti-**CCR5** antibody of claim 1, wherein the heavy chains are expressed by the plasmid designated pVg1:HuPRO140 HG2-VH (ATCC Deposit Designation PTA-4098).

3. The anti-**CCR5** antibody of claim 1, wherein the heavy chains are expressed by the plasmid designated pVg1:HuPRO140 (mut B+D+I)-VH (ATCC Deposit Designation PTA-4099).

4. An anti-**CCR5** antibody comprising two light chains, each chain comprising consecutive amino acids, the amino acid sequence of which is set forth in SEQ ID NO: 6, and two heavy chains, each heavy chain comprising consecutive amino acids, the amino acid sequence of which is set forth in SEQ ID NO: 9.

5. An anti-**CCR5** antibody comprising two light chains, each light chain comprising consecutive amino acids, the amino acid sequence of which is set forth in SEQ ID NO: 6, and two heavy chains, each heavy chain comprising consecutive amino acids, the amino acid sequence of which is set forth in SEQ ID NO: 12.

6. An isolated nucleic acid encoding a polypeptide comprising consecutive amino acids, the amino acid sequence of which is set forth in SEQ ID NO: 6.

7. The nucleic acid of claim 6, wherein the consecutive amino acids are the amino acids expressed by a plasmid designated pVK:HuPRO140-VK (ATCC Deposit Designation PTA-4097).

8. The nucleic acid of claim 6, wherein the nucleic acid comprises the sequence set forth in SEQ ID NO: 5.

9. The nucleic acid of any one of claims 6, 7 or 8, wherein the nucleic acid is RNA, DNA or cDNA.

10. An isolated nucleic acid encoding a polypeptide comprising consecutive amino acids, the amino acid sequence of which is set forth in SEQ ID NO: 9.

11. The nucleic acid of claim 10, wherein the consecutive amino acids are the amino acids expressed by a plasmid designated pVg1:HuPRO140 HG2-VH (ATCC Deposit Designation PTA-4098).

12. The nucleic acid of claim 10, wherein the nucleic acid comprises the sequence set forth in SEQ ID NO: 8.

13. The nucleic acid of any one of claims 10, 11 or 12 wherein the nucleic acid is RNA, DNA or cDNA.

14. An isolated nucleic acid encoding a polypeptide comprising consecutive amino acids, the amino acid sequence of which is set forth in SEQ ID NO: 12.

15. The nucleic acid of claim 14, wherein the consecutive amino acids are the amino acids expressed by a plasmid designated pVg1:HuPRO140 (mut B+D+I)-VH (ATCC Deposit Designation PTA-4099)

16. The nucleic acid of claim 14, wherein the nucleic acid comprises the

17. The nucleic acid of any one of claims 14, 15 and 16, wherein the nucleic acid is RNA, DNA or cDNA.

18. A composition comprising at least one of the anti-**CCR5** antibody or a fragment thereof, of any one of claims 1-5 and a carrier.

19. A composition comprising the anti-**CCR5** antibody or a fragment thereof, of any one of claims 1-5, having attached thereto a material selected from the group consisting of a radioisotope, a toxin, polyethylene glycol, a cytotoxic agent and a detectable label.

20. A method of inhibiting HIV-1 infection of a CD4+ cell which comprises contacting the CD4+ cell with an antibody which comprises (i) two light chains, each light chain comprising the expression product of a plasmid designated pVK:HuPRO140-VK (ATCC Deposit Designation PTA-4097), and (ii) two heavy chains, each heavy chain comprising the expression product of either a plasmid designated pVg1:HuPRO140 HG2-VH (ATCC Deposit Designation PTA-4098) or a plasmid designated pVg1:HuPRO140 (mut B+D+I)-VH (ATCC Deposit Designation PTA-4099), or a fragment of such antibody which binds to **CCR5** on the surface of the CD4+ cell, in an amount and under conditions such that fusion of HIV-1 or an HIV-1 infected cell to-the CD4+ cell is inhibited, thereby inhibiting HIV-1 infection of the CD4+ cell.

21. The method of claim 20, wherein the CD4+ cell expresses **CCR5**.

22. A method of treating a subject afflicted with HIV-1 which comprises administering to the subject an effective HIV-1 treating dosage amount of an anti-**CCR5** antibody comprising (i) two light chains, each light chain comprising the expression product of a plasmid designated pVK:HuPRO140-VK (ATCC Deposit Designation PTA-4097), and (ii) two heavy chains, each heavy chain comprising the expression product of either a plasmid designated pVg1:HuPRO140 HG2-VH (ATCC Deposit Designation PTA-4098) or a plasmid designated pVg1:HuPRO140 (mut B+D+I)-VH (ATCC Deposit Designation PTA-4099), or a fragment of such antibody, which binds to **CCR5** on the surface of a human cell, under conditions effective to treat said HIV-1-afflicted subject.

23. A method of preventing a subject from contracting an HIV-1 infection which comprises administering to the subject an effective HIV-1 infection-preventing dosage amount of an anti-**CCR5** antibody comprising (i) two light chains, each light chain comprising the expression product of a plasmid designated pVK:HuPRO140-VK (ATCC Deposit Designation PTA-4097), and (ii) two heavy chains, each heavy chain comprising the expression product of either a plasmid designated pVg1:HuPRO 140 HG2-VH (ATCC Deposit Designation PTA-4098) or a plasmid designated pVg1:HuPRO140 (mut B+D+I)-VH (ATCC Deposit Designation PTA-4099), or a fragment of such antibody, which binds to **CCR5** on the surface of a human cell, under conditions effective to prevent said HIV-1 infection in said subject.

24. The method of claim 22 or 23, wherein the anti-**CCR5** antibody is administered to the subject by a method selected from the group consisting of intravenous, intramuscular and subcutaneous means.

25. The method of claim 22 or 23, wherein the anti-**CCR5** antibody is administered continuously to said subject.

26. The method of claim 22 or 23 wherein the anti-**CCR5** antibody is administered at predetermined periodic intervals to said subject.

27. The method of claim 22 or 23, which further comprises labeling the anti-**CCR5** antibody with a detectable marker.

28. The method of claim 27, wherein the detectable marker is a radioactive or a fluorescent marker.

29. The method of claim 22 or 23, wherein the dosage of said anti-**CCR5** antibody ranges from about 0.1 to about 100,000 μg/kg body weight of said subject.

30. The method of claim 29, wherein the dosage of said anti-**CCR5** antibody does not inhibit an endogenous chemokine activity on **CCR5** in said subject.

31. An anti-**CCR5** antibody conjugate comprising an anti-**CCR5** antibody which comprises (i) two light chains, each light chain comprising the expression product of a plasmid designated pVK:HuPRO140-VK (ATCC Deposit Designation PTA-4097), and (ii) two heavy chains, each heavy chain comprising the expression product of either a plasmid designated pVg1:HuPRO140 HG2-VH (ATCC Deposit Designation PTA-4098) or a plasmid

PTA-4099), or a fragment of such antibody which binds to **CCR5** on the surface of a human cell, conjugated to at least one polymer.

32. The anti-**CCR5** antibody conjugate of claim 31, wherein the polymer is selected from the group consisting of hydrophilic polyvinyl polymers, polyalkylene ethers, polyoxyalkylenes, polymethacrylates, carbomers, branched polysaccharides, unbranched polysaccharides, polymers of sugar alcohols, heparin and heparon.

33. The anti-**CCR5** antibody conjugate of claim 32, wherein the polyalkylene ether is polyethylene glycol (PEG) or a derivative thereof.

34. The anti-**CCR5** antibody conjugate of claim 33, wherein at least one PEG has an average molecular weight of at least 20 kD.

35. The anti-**CCR5** antibody conjugate of claim 31, wherein the apparent size of the conjugate is at least about 500 kD.

36. The anti-**CCR5** antibody conjugate of claim 31, wherein the conjugate has at least one of an increase in serum half-life, an increase in mean residence time in the circulation and a decrease in serum clearance rate, compared to a nonconjugated anti-**CCR5** antibody or fragment thereof.

37. A method of inhibiting infection of a **CCR5**+ cell by HIV-1, which method comprises administering to a subject at risk of HIV-1 infection the conjugate of claim 31 in an amount and under conditions effective to inhibit infection of **CCR5**+ cells of said subject by HIV-1.

38. A method of treating an HIV-1 infection in a subject, which method comprises administering to an HIV-1-infected subject the conjugate of claim 31 in an amount and under conditions effective to treat the subject's HIV-1 infection.

39. The method of claim 38, wherein the amount of the conjugate is effective in reducing a viral load in the subject.

40. The method of claim 38, wherein the amount of the conjugate is effective in increasing a CD4+ cell count in the subject.

41. The method of claim 38, which further comprises administering to said subject at least one conventional anti-viral agent.

42. The method of claim 37 or 38, wherein the conjugate is administered to the subject by a method selected from the group consisting of intravenous, intramuscular and subcutaneous means.

43. The method of claim 37 or 38, wherein the conjugate is administered continuously to said subject.

44. The method of claim 37 or 38, wherein the conjugate is administered at predetermined periodic intervals to said subject.

45. The method of claim 37 or 38, which further comprises labeling the conjugate with a detectable marker.

46. The method of claim 45, wherein the detectable marker is a radioactive or a fluorescent marker.

47. A transformed host cell comprising at least two vectors, at least one vector comprising a nucleic acid sequence encoding heavy chains of an anti-**CCR5** antibody, and at least one vector comprising a nucleic acid sequence encoding light chains of the anti-**CCR5** antibody, wherein the anti-**CCR5** antibody comprises two heavy chains having the amino acid sequence set forth in SEQ ID NO: 9, and two light chains having the amino acid sequence set forth in SEQ ID NO: 6.

48. A transformed host cell comprising at least two vectors, at least one vector comprising a nucleic acid sequence encoding heavy chains of an anti-**CCR5** antibody, and at least one vector comprising a nucleic acid sequence encoding light chains of the anti-**CCR5** antibody, wherein the anti-**CCR5** antibody comprises two heavy chains having the amino acid sequence set forth in SEQ ID NO: 12, and two light chains having the amino acid sequence set forth in SEQ ID NO: 6.

49. The transformed host cell of claim 47 or 48, wherein the cell is a mammalian cell.

50. The transformed host cell of claim 49 wherein the cell is a COS cell, a CHO cell or a myeloma cell.

51. The transformed host cell of claim 47 or 48, wherein the cell

52. The transformed host cell of claim 47, wherein the vector encoding heavy chains is designated pVg1:HuPRO140 HG2-VH (ATCC Deposit Designation PTA-4098).

53. The transformed host cell of claim 48, wherein the vector encoding heavy chains is designated pVg1:HuPRO140 (mut B+D+I)-VH (ATCC Deposit Designation PTA-4099).

54. The transformed host cell of claim 47 or 48, wherein the vector encoding light chains is designated pVK:HuPRO140-VK (ATCC Deposit Designation PTA-4097).

55. The transformed host cell of claim 47, wherein the vector encoding heavy chains is designated pVg1:HuPRO140 HG2-VH (ATCC Deposit Designation PTA-4098) and the vector encoding light chains is designated pVK:HuPRO140-VK (ATCC Deposit Designation PTA-4097).

56. The transformed host cell of claim 48, wherein the vector encoding the heavy chains is designated pVg1:HuPRO140 (mut B+D+I)-VH (ATCC Deposit Designation PTA-4099) and the vector encoding light chains is designated pVK-HuPRO140-VK (ATCC Deposit Designation PTA-4097).

57. The transformed host cell of claim 47, wherein the nucleic acid sequence encoding heavy chains has the nucleic acid sequence set forth in SEQ. ID NO: 8.

58. The transformed host cell of claim 48, wherein the nucleic acid sequence encoding heavy chains has the nucleic acid sequence set forth in SEQ ID NO: 11.

59. The transformed host cell of claim 47 or 48 wherein the nucleic acid sequence encoding light chains has the nucleic acid sequence set forth in SEQ ID NO: 5.

60. A vector comprising a nucleic acid sequence encoding a heavy chain of an anti-**CCR5** antibody, wherein the heavy chain comprises the amino acid sequence set forth in SEQ ID NO: 9.

61. The vector of claim 60, wherein the vector is designated pVg1:HuPRO140 HG2-VH (ATCC Deposit Designation No. PTA-4098).

62. A vector comprising a nucleic acid sequence encoding a heavy chain of an anti-**CCR5** antibody, wherein the heavy chain comprises the amino acid sequence set forth in SEQ ID NO: 12.

63. The vector of claim 62, wherein the vector is designated pVg1:HuPRO140 (mut B+D+I)-VH (ATCC Deposit Designation No. PTA-4099).

64. A vector comprising a nucleic acid sequence encoding a light chain of an anti-**CCR5** antibody, wherein the light chain comprises the amino acid sequence set forth in SEQ ID NO: 6.

65. The vector of claim 64, wherein the vector is designated pVK:HuPRO140-VK (ATCC Deposit Designation No. PTA-4097).

66. A process for producing an anti-**CCR5** antibody which comprises culturing a host cell containing therein (i) a plasmid designated pVK:HuPRO140-VK (ATCC Deposit Designation PTA-4097), and (ii) either a plasmid designated pVg1:HuPRO140 HG2-VH (ATCC Deposit Designation PTA-4098) or a plasmid designated pVg1:PRO140 (mut B+D+I)-VH (ATCC Deposit Designation PTA-4099) under conditions permitting the production of an antibody comprising two light chains encoded by the plasmid designated pVK:HuPRO140 HG2-VH (ATCC Deposit Designation PTA-4097)and two heavy chains encoded either by the plasmid designated pVg1:HuPRO140 HG2-VH (ATCC Deposit Designation PTA-4098) or by the plasmid designated pVg1:HuPRO 140 (mut B+D+I)-VH (ATCC Deposit Designation PTA-4099), so as to thereby produce an anti-**CCR5** antibody.

67. A process for producing an anti-**CCR5** antibody which comprises: a) transforming a host cell with (i) a plasmid designated pVK:HuPRO140-VK (ATCC Deposit Designation PTA-4097), and (ii) either a plasmid designated pVg1:HuPRO140 HG2-VH (ATCC Deposit Designation PTA-4098) or a plasmid designated pVg1:HuPRO140 (mut B+D+I)-VH (ATCC Deposit Designation PTA-4099); and b) culturing the transformed host cell under conditions permitting production of an antibody comprising two light chains encoded by the plasmid designated pVK:HuPRO140-VK (ATCC Deposit Designation PTA-4097) and two heavy chains encoded either by the plasmid designated pVg1:HuPRO140 HG2-VH (ATCC Deposit Designation PTA-4098) or by the plasmid designated pVg1:HuPRO140 (mut B+D+I)-VH (ATCC Deposit Designation PTA-4099), so as to thereby produce an anti-**CCR5** antibody.

anti-**CCR5** antibody so produced in isolated form.

69. The method of claim 66 or 67, wherein the host cell is a mammalian cell.

70. The method of claim 69, wherein the mammalian host cell is a COS cell, a CHO cell or a myeloma cell.

71. The method of claim 66 or 67, wherein the heavy chains of the anti-**CCR5** antibody are encoded by the plasmid designated pVg1:HuPRO140 HG2-VH (ATCC Deposit Designation PTA-4098).

72. The method of claim 66 or 67, wherein the heavy chains of the anti-**CCR5** antibody are encoded by the plasmid designated pVg1:HuPRO140 (mut B+D+I) (ATCC Deposit Designation PTA-4099).

73. A kit for use in a process of producing an anti-**CCR5** antibody comprising: a) a vector comprising a nucleic acid sequence encoding a light chain of an anti-**CCR5** antibody, wherein the light chain comprises the amino acid sequence set forth in SEQ ID NO: 6; and b) a vector comprising a nucleic acid sequence encoding a heavy chain of an anti-**CCR5** antibody, wherein the heavy chain comprises the amino acid sequence set forth in SEQ ID NO: 9, or a vector comprising a nucleic acid sequence encoding a heavy chain of an anti-**CCR5** antibody, wherein the heavy chain comprises the amino acid sequence set forth in SEQ ID NO: 12.

L6 ANSWER 7 OF 11 USPATFULL on STN
2003:119700 Compositions and methods for inhibition of hiv-1 infection.
Olson, William C., Ossining, NY, UNITED STATES
Maddon, Paul J., Scarsdale, NY, UNITED STATES
US 2003082185 A1 20030501
APPLICATION: US 2000-493346 A1 20000128 (9)
DOCUMENT TYPE: Utility; APPLICATION.
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
CLM What is claimed is:

1. A composition which comprises an admixture of two compounds, wherein one compound retards attachment of HIV-1 to a CD4+ cell by retarding binding of HIV-1 gp120 envelope glycoprotein to CD4 on the surface of the CD4+ cell and the other compound retards gp41 from adopting a conformation capable of mediating fusion of HIV-1 to a CD4+ cell by binding noncovalently to an epitope on a gp41 fusion intermediate, wherein the relative mass ratio of the compounds in the admixture ranges from about 100:1 to about 1:100, the composition being effective to inhibit HIV-1 infection of the CD4+ cell.

2. The composition of claim 1, wherein the compound which retards attachment of HIV-1 to the CD4+ cell by retarding binding of HIV-1 gp120 envelope glycoprotein to CD4 on the surface of the CD4+ cell is a CD4-based protein.

3. The composition of claim 2, wherein the CD4-based protein is a CD4-immunoglobulin fusion protein.

4. The composition of claim 3, wherein the CD4-immunoglobulin fusion protein is CD4-IgG2, wherein the CD4-IgG2 comprises two heavy chains and two lights chains, wherein the heavy chains are encoded by an expression vector designated CD4-IgG2HC-pRcCMV (ATCC Accession No. 75193) and the light chains are encoded by an expression vector designated CD4-kLC-pRcCMV (ATCC Accession No. 75194).

5. The composition of claim 1, wherein the compound which retards attachment of HIV-1 to the CD4+ cell by retarding binding of HIV-1 gp120 envelope glycoprotein to CD4 on the surface of the CD4+ cell is a protein, the amino acid sequence of which comprises that of a protein found in HIV-1 as an envelope glycoprotein.

6. The composition of claim 5, wherein the protein binds to an epitope of CD4 on the surface of the CD4+ cell.

7. The composition of claim 6, wherein the envelope glycoprotein is selected from the group consisting of gp120, gp160, and gp140.

8. The composition of claim 1, wherein the compound which retards the attachment of HIV-1 to the CD4+ cell by retarding binding of HIV-1 gp120 envelope glycoprotein to CD4 on the surface of the CD4+ cell is an antibody or portion of an antibody.

9. The composition of claim 8, wherein the antibody is a monoclonal antibody.

human, humanized or chimeric antibody.

11. The composition of claim 8, wherein the portion of the antibody is a Fab fragment of the antibody.

12. The composition of claim 8, wherein the portion of the antibody comprises the variable domain of the antibody.

13. The composition of claim 8, wherein the portion of the antibody comprises a CDR portion of the antibody.

14. The composition of claim 9, wherein the monoclonal antibody is an IgG, IgM, IgD, IgA, or IgE monoclonal antibody.

15. The composition of claim 9, wherein the monoclonal antibody binds to an HIV-1 envelope glycoprotein.

16. The composition of claim 15, wherein the HIV-1 envelope glycoprotein is selected from the group consisting of gp120 and gp160.

17. The composition of claim 16, wherein HIV-1 envelope glycoprotein is gp120 and the monoclonal antibody which binds to gp120 is IgG1b12 or F105.

18. The composition of claim 8, wherein the antibody binds to an epitope of CD4 on the surface of the CD4+ cell.

19. The composition of claim 1, wherein the compound which retards attachment of HIV-1 to the CD4+ cell by retarding binding of HIV-1 gp120 envelope glycoprotein to CD4 on the surface of the CD4+ cell is a peptide.

20. The composition of claim 1, wherein the compound which retards attachment of HIV-1 to the CD4+ cell by retarding binding of HIV-1 gp120 envelope glycoprotein to CD4 on the surface of the CD4+ cell is a nonpeptidyl agent.

21. The composition of claim 1, wherein the compound which retards gp41 from adopting a conformation capable of mediating fusion of HIV-1 to a CD4+ cell by binding noncovalently to an epitope on a gp41 fusion intermediate is an antibody.

22. The composition of claim 21, wherein the antibody is a monoclonal antibody.

23. The composition of claim 1, wherein the compound which retards gp41 from adopting a conformation capable of mediating fusion of HIV-1 to a CD4+ cell by binding noncovalently to an epitope on a gp41 fusion intermediate is a peptide.

24. The composition of claim 1, wherein the compound which retards gp41 from adopting a conformation capable of mediating fusion of HIV-1 to a CD4+ cell by binding noncovalently to an epitope on a gp41 fusion intermediate is a fusion protein which comprises a peptide selected from the group consisting of T-20 (SEQ ID NO: 1), DP107 (SEQ ID NO: 2), N34 (SEQ ID NO: 3), C28 (SEQ ID NO: 4), and N34(L6)C28 (SEQ ID NO: 5).

25. The composition of claim 23, wherein the peptide is selected from the group consisting of T-20 (SEQ ID NO: 1), DP107 (SEQ ID NO: 2), N34 (SEQ ID NO: 3), C28 (SEQ ID NO: 4), and N34(L6)C28 (SEQ ID NO: 5).

26. The composition of claim 23, wherein the peptide is T-20 (SEQ ID NO: 1).

27. The composition of claim 1, wherein the compound which retards gp41 from adopting a conformation capable of mediating fusion of HIV-1 to a CD4+ cell by binding noncovalently to an epitope on a gp41 fusion intermediate is a non-peptidyl agent.

28. The composition of claim 1, wherein the relative mass ratio of each such compound in the admixture ranges from about 25:1 to about 1:1.

29. The composition of claim 28, wherein the mass ratio is about 25:1

30. The composition of claim 28, wherein the mass ratio is about 5:1.

31. The composition of claim 28, wherein the mass ratio is about 1:1.

32. The composition of claim 1, wherein the composition is admixed with a carrier.

33. The composition of claim 32, wherein the carrier is an aerosol,

34. A method of inhibiting HIV-1 infection of a CD4+ cell which comprises contacting the CD4+ cell with an amount of the composition of claim 1 effective to inhibit HIV-1 infection of the CD4+ cell so as to thereby inhibit HIV-1 infection of the CD4+ cell.

35. The method of claim 34, wherein the CD4+ cell is present in a subject and the contacting is effected by administering the composition to the subject.

36. The method of claim 33, wherein the effective amount of the composition comprises from about 0.000001 mg/kg body weight to about 100 mg/kg body weight of the subject.

37. A method of inhibiting HIV-1 infection of a CD4+ cell which comprises contacting the CD4+ cell with an amount of a compound which retards attachment of HIV-1 to the CD4+ cell by retarding binding of HIV-1 gp120 envelope glycoprotein to CD4 on the surface of the CD4+ cell effective to inhibit HIV-1 infection of the CD4+ cell and an amount of a compound which retards gp41 from adopting a conformation capable of mediating fusion of HIV-1 to a CD4+ cell by binding noncovalently to an epitope on a gp41 fusion intermediate so as to thereby inhibit HIV-1 infection of the CD4+ cell.

38. The method of claim 37, wherein the CD4+ cell is present in a subject and the contacting is effected by administering the compounds to the subject.

39. The method of claim 38, wherein the compounds are administered to the subject simultaneously.

40. The method of claim 38, wherein the compounds are administered to the subject at different times.

41. The method of claim 38, wherein the compounds are administered to the subject by different routes of administration.

L6 ANSWER 9 OF 11 USPATFULL on STN
2003:64291 Methods for inhibiting HIV-1 infection.
Olson, William C., Ossining, NY, UNITED STATES
Maddon, Paul J., Scarsdale, NY, UNITED STATES
US 2003044411 A1 20030306
APPLICATION: US 2002-116797 A1 20020405 (10)
PRIORITY: US 2001-282380P 20010406 (60)
DOCUMENT TYPE: Utility; APPLICATION.
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
CLM What is claimed is:
1. A method of reducing an HIV infected subject's HIV-1 viral load which comprises administering to the subject an effective viral load reducing amount of an antibody which (a) binds to a **CCR5** chemokine receptor and (b) inhibits fusion of HIV-1 to a CD4+**CCR5**+ cell, so as to thereby reduce the subject's HIV-1 viral load to 50% or less of the subject's HIV-1 viral load prior to administering the antibody to the subject.

2. The method of claim 1, wherein the antibody is a monoclonal antibody.

3. The method of claim 1, wherein the antibody is selected from the group consisting of PA8 (ATCC Accession No. HB-12605), PA9 (ATCC Accession No. HB-12606), PA10 (ATCC Accession No.HB-12607), PA11 (ATCC Accession No. HB-12608), PA12 (ATCC Accession No. HB-12609), and PA14 (ATCC Accession No. HB12610).

4. The method of claim 1, wherein the antibody is PA14 (ATCC Accession No. HB-12610).

5. The method of claim 1, wherein the subject's HIV-1 viral load is reduced to 33% or less of the subject's HIV-1 viral load prior to administering the antibody to the subject.

6. The method of claim 1, wherein the subject's HIV-1 viral load is reduced to 10% or less of the subject's HIV-1 viral load prior to administering the antibody to the subject.

7. The method of claim 1, wherein the reduction of the subject's HIV-1 viral load is sustained for a period of time.

8. The method of claim 7, wherein the period of time is at least one day.

9. The method of claim 7, wherein the period of time is at least three days.

10. The method of claim 7, wherein the period of time is at least seven days.

11. The method of claim 1, wherein the effective amount of the antibody is between about 1 mg and about 50 mg per kg body weight of the subject.

12. The method of claim 11, wherein the effective amount of the antibody is between about 2 mg and about 40 mg per kg body weight of the subject.

13. The method of claim 12, wherein the effective amount of the antibody is between about 3 mg and about 30 mg per kg body weight of the subject.

14. The method of claim 13, wherein the effective amount of the antibody is between about 4 mg and about 20 mg per kg body weight of the subject.

15. The method of claim 14, wherein the effective amount of the antibody is between about 5 mg and about lomg per kg body weight of the subject.

16. The method of claim 1, wherein the antibody is administered at least once per day.

17. The method of claim 1, wherein the antibody is administered daily.

18. The method of claim 1, wherein the antibody is administered every other day.

19. The method of claim 1, wherein the antibody is administered every 6 to 8 days.

20. The method of claim 1, wherein the antibody is administered weekly.

21. The method of claim 1, wherein the antibody is administered intravenously, subcutaneously, intramuscularly, intraperitoneally, orally or topically.

22. The method of claim 1, wherein the subject is a human being and the antibody is a humanized antibody.

23. Use of an effective amount of the antibody which (a) binds to a **CCR5** chemokine receptor and (b) inhibits fusion of HIV-1 to a CD4+**CCR5**+ cell for the preparation of a pharmaceutical composition to reduce a subject's HIV-1 viral load.

24. The use of claim 23, wherein the antibody is a monoclonal antibody.

25. The use of claim 23, wherein the antibody is selected from the group consisting of PA8 (ATCC Accession No. HB-12605), PA9 (ATCC Accession No. HB-12606), PA10 (ATCC Accession No.HB-12607), PA11 (ATCC Accession No. HS-12608), PA12 (ATCC Accession No. HB-12609), and PA14 (ATCC Accession No. HB-12610).

26. The use of claim 23, wherein the antibody is PA14 (ATCC Accession No. HB-12610).

27. The use of claim 23, wherein the effective amount of the antibody is between about 1 mg and about 50 mg per kg body weight of the subject.

28. The use of claim 27, wherein the effective amount of the antibody is between about 2 mg and about 40 mg per kg body weight of the subject.

29. The use of claim 28, wherein the effective amount of the antibody is between about 3 mg and about 30 mg per kg body weight of the subject.

30. The use of claim 29, wherein the effective amount of the antibody is between about 4 mg and about 20 mg per kg body weight of the subject.

31. The use of claim 30, wherein the effective amount of the antibody is between about 5 mg and about 10 mg per kg body weight of the subject.

32. The use of any one of claims 23-31, wherein the preparation of the pharmaceutical composition comprises admixing the effective amount of the antibody and a pharmaceutically acceptable carrier.

L6 ANSWER 10 OF 11 USPATFULL on STN
2002:265543 Methods for inhibiting HIV-1 infection.
Olson, William C., Ossining, NY, UNITED STATES
Maddon, Paul J., Scarsdale, NY, UNITED STATES
US 2002146415 A1 20021010
APPLICATION: US 2001-828615 A1 20010406 (9)
DOCUMENT TYPE: Utility; APPLICATION.
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

1. A method of reducing an HIV infected subject's HIV-1 viral load which comprises administering to the subject an effective viral load reducing amount of an antibody which (a) binds to a **CCR5** chemokine receptor and (b) inhibits fusion of HIV-1 to a CD4+**CCR5**+ cell, so as to thereby reduce the subject's HIV-1 viral load to 50% or less of the subject's HIV-1 viral load prior to administering the antibody to the subject.

2. The method of claim 1, wherein the antibody is a monoclonal antibody.

3. The method of claim 1, wherein the antibody is selected from the group consisting of PA8 (ATCC Accession No. HB-12605), PA9(ATCC Accession No. HB-12606), PA10 (ATCC Accession No. HB-12607), PA11 (ATCC Accession No. HB-12608), PA12 (ATCC Accession No. HB-12609), and PA14 (ATCC Accession No. HB-12610).

4. The method of claim 1, wherein the antibody is PA14 (ATCC Accession No. HB-1261).

5. The method of claim 1, wherein the subject's HIV-1 viral load is reduced to 33% or less of the subject's HIV-1 viral load prior to administering the antibody to the subject.

6. The method of claim 1, wherein the subject's HIV-1 viral load is reduced to 10% or less of the subject's HIV-1 viral load prior to administering the antibody to the subject.

7. The method of claim 1, wherein the reduction of the subject's HIV-1 viral load is sustained for a period of time.

8. The method of claim 7, wherein the period of time is at least one day.

9. The method of claim 7, wherein the period of time is at least three days.

10. The method of claim 7, wherein the period of time is at least seven days.

11. The method of claim 1, wherein the effective amount of the antibody is between about 1 mg and about 50 mg per kg body weight of the subject.

12. The method of claim 11, wherein the effective amount of the antibody is between about 2 mg and about 40 mg per kg body weight of the subject.

13. The method of claim 12, wherein the effective amount of the antibody is between about 3 mg and about 30 mg per kg body weight of the subject.

14. The method of claim 13, wherein the effective amount of the antibody is between about 4 mg and about 20 mg per kg body weight of the subject.

15. The method of claim 14, wherein the effective amount of the antibody is between about 5 mg and about 10 mg per kg body weight of the subject.

16. The method of claim 1, wherein the antibody is administered at least once per day.

17. The method of claim 1, wherein the antibody is administered daily.

18. The method of claim 1, wherein the antibody is administered every other day.

19. The method of claim 1, wherein the antibody is administered every 6 to 8 days.

20. The method of claim 1, wherein the antibody is administered weekly.

21. The method of claim 1, wherein the antibody is administered intravenously, subcutaneously, intramuscularly, intraperitoneally, orally or topically.

22. The method of claim 1, wherein the subject is a human being and the antibody is a humanized antibody.

L6 ANSWER 11 OF 11 USPATFULL on STN
2002:198280 Compositions and methods for inhibition of HIV-1 infection.
Olson, William C., Ossining, NY, UNITED STATES
Maddon, Paul J., Scarsdale, NY, UNITED STATES
US 2002106374 A1 20020808
APPLICATION: US 2001-912824 A1 20010725 (9)
PRIORITY: US 2001-266738P 20010206 (60)
DOCUMENT TYPE: Utility; APPLICATION.

CLM What is claimed is:

1. A composition which comprises an admixture of two compounds, wherein: (a) one compound is an antibody or portion thereof which binds to a **CCR5** receptor; and (b) one compound retards gp41 from adopting a conformation capable of mediating fusion of HIV-1 to a CD4+ cell by binding noncovalently to an epitope on a gp41 fusion intermediate; wherein the relative mass ratio of the compounds in the admixture ranges from about 100:1 to about 1:100, the composition being effective to inhibit HIV-1 infection of the CD4+ cell.

2. A composition which comprises an admixture of three compounds, wherein: (a) one compound is an antibody or portion thereof which binds to a **CCR5** receptor; (b) one compound retards attachment of HIV-1 to a CD4+ cell by retarding binding of HIV-1 gp120 envelope glycoprotein to CD4 on the surface of the CD4+ cell; and (c) one compound retards gp41 from adopting a conformation capable of mediating fusion of HIV-1 to a CD4+ cell by binding noncovalently to an epitope on a gp41 fusion intermediate; wherein the relative mass ratio of any two of the compounds in the admixture ranges from about 100:1 to about 1:100, the composition being effective to inhibit HIV-1 infection of the CD4+ cell.

3. The composition of claim 2, wherein the compound which retards attachment of HIV-1 to the CD4+ cell by retarding binding of HIV-1 gp120 envelope glycoprotein to CD4 on the surface of the CD4+ cell is a CD4-based protein.

4. The composition of claim 3, wherein the CD4-based protein is a CD4-immunoglobulin fusion protein.

5. The composition of claim 4, wherein the CD4-immunoglobulin fusion protein is CD4-IgG2, wherein the CD4-IgG2 comprises two heavy chains and two lights chains, wherein the heavy chains are encoded by an expression vector designated CD4-IgG2HC-pRcCMV (ATCC Accession No. 75193) and the light chains are encoded by an expression vector designated CD4-kLC-pRcCMV (ATCC Accession No. 75194).

6. The composition of claim 2, wherein the compound which retards attachment of HIV-1 to the CD4+ cell by retarding binding of HIV-1 gp120 envelope glycoprotein to CD4 on the surface of the CD4+ cell is a protein, the amino acid sequence of which comprises that of a protein found in HIV-1 as an envelope glycoprotein.

7. The composition of claim 6, wherein the protein binds to an epitope of CD4 on the surface of the CD4+ cell.

8. The composition of claim 7, wherein the envelope glycoprotein is selected from the group consisting of gp120, gp160, and gp140.

9. The composition of claim 2, wherein the compound which retards the attachment of HIV-1 to the CD4+ cell by retarding binding of HIV-1 gp120 envelope glycoprotein to CD4 on the surface of the CD4+ cell is an antibody or portion of an antibody.

10. The composition of claim 9, wherein the antibody is a monoclonal antibody.

11. The composition of claim 10, wherein the monoclonal antibody is a human, humanized or chimeric antibody.

12. The composition of claim 9, wherein the portion of the antibody is a Fab fragment of the antibody.

13. The composition of claim 9, wherein the portion of the antibody comprises the variable domain of the antibody.

14. The composition of claim 9, wherein the portion of the antibody comprises a CDR portion of the antibody.

15. The composition of claim 10, wherein the monoclonal antibody is an IgG, IgM, IgD, IgA, or IgE monoclonal antibody.

16. The composition of claim 10, wherein the monoclonal antibody binds to an HIV-1 envelope glycoprotein.

17. The composition of claim 16, wherein the HIV-1 envelope glycoprotein is selected from the group consisting of gp120 and gp160.

18. The composition of claim 16, wherein HIV-1 envelope glycoprotein is gp120 and the monoclonal antibody which binds to gp120 is IgG1b12 or F105.

19. The composition of claim 9, wherein the antibody binds to an epitope

20. The composition of claim 2, wherein the compound which retards attachment of HIV-1 to the CD4+ cell by retarding binding of HIV-1 gp120 envelope glycoprotein to CD4 on the surface of the CD4+ cell is a peptide.

21. The composition of claim 2, wherein the compound which retards attachment of HIV-1 to the CD4+ cell by retarding binding of HIV-1 gp120 envelope glycoprotein to CD4 on the surface of the CD4+ cell is a nonpeptidyl agent.

22. The composition of claim 1 or 2, wherein the compound which retards gp41 from adopting a conformation capable of mediating fusion of HIV-1 to a CD4+ cell by binding noncovalently to an epitope on a gp41 fusion intermediate is an antibody.

23. The composition of claim 22, wherein the antibody is a monoclonal antibody.

24. The composition of claim 1 or 2, wherein the compound which retards gp41 from adopting a conformation capable of mediating fusion of HIV-1 to a CD4+ cell by binding noncovalently to an epitope on a gp41 fusion intermediate is a peptide.

25. The composition of claim 1 or 2, wherein the compound which retards gp41 from adopting a conformation capable of mediating fusion of HIV-1 to a CD4+ cell by binding noncovalently to an epitope on a gp41 fusion intermediate is a fusion protein which comprises a peptide selected from the group consisting of T-20 (SEQ ID NO: 1), DP107 (SEQ ID NO: 2), N34 (SEQ ID NO: 3), C28 (SEQ ID NO: 4), N34(L6)C28 (SEQ ID NO: 5), and T-1249 (SEQ ID NO:6).

26. The composition of claim 24, wherein the peptide is selected from the group consisting of T-20 (SEQ ID NO: 1), DP107 (SEQ ID NO: 2), N34 (SEQ ID NO: 3), C28 (SEQ ID NO: 4), N34(L6)C28 (SEQ ID NO: 5), and T-1249 (SEQ ID NO:6).

27. The composition of claim 24, wherein the peptide is T-20 (SEQ ID NO: 1).

28. The composition of claim 1 or 2, wherein the compound which retards gp41 from adopting a conformation capable of mediating fusion of HIV-1 to a CD4+ cell by binding noncovalently to an epitope on a gp41 fusion intermediate is a non-peptidyl agent.

29. The composition of claim 1 or 2, wherein the antibody which binds to a **CCR5** receptor is selected from the group consisting of PA8 (ATCC Accession No. HB-12605), PA10 (ATCC Accession No.12607), PA11 (ATCC Accession No. HB-12608), PA12 (ATCC Accession No. HB-12609), and PA14 (ATCC Accession No. HB-12610).

30. The composition of claim 1 or 2, wherein the antibody is PA14 (ATCC Accession No. HB-12610).

31. The composition of claim 29, wherein the antibody is a monoclonal antibody.

32. The composition of claim 29, wherein the monoclonal antibody is a human, humanized or chimeric antibody.

33. The composition of claim 1 or 2, wherein the portion of the antibody is a Fab fragment of the antibody.

34. The composition of claim 1 or 2, wherein the portion of the antibody comprises the variable domain of the antibody.

35. The composition of claim 1 or 2, wherein the portion of the antibody comprises a CDR portion of the antibody.

36. The composition of claim 31, wherein the monoclonal antibody is an IgG, IgM, IgD, IgA, or IgE monoclonal antibody.

37. The composition of claim 1 or 2, wherein the relative mass ratio of each such compound in the admixture ranges from about 25:1 to about 1:1.

38. The composition of claim 37, wherein the mass ratio is about 25:1

39. The composition of claim 37, wherein the mass ratio is about 5:1.

40. The composition of claim 37, wherein the mass ratio is about 1:1.

41. The composition of claim 1 or 2, wherein the composition is admixed

42. The composition of claim 41, wherein the carrier is an aerosol, intravenous, oral or topical carrier.

43. A method of inhibiting HIV-1 infection of a CD4+ cell which comprises contacting the CD4+ cell with an amount of the composition of claim 1 or 2 effective to inhibit HIV-1 infection of the CD4+ cell so as to thereby inhibit HIV-1 infection of the CD4+ cell.

44. The method of claim 43, wherein the CD4+ cell is present in a subject and the contacting is effected by administering the composition to the subject.

45. The method of claim 43, wherein the effective amount of the composition comprises from about 0.000001 mg/kg body weight to about 100 mg/kg body weight of the subject.

46. A method of inhibiting HIV-1 infection of a CD4+ cell which comprises contacting the CD4+ cell with (1) an amount of an antibody which binds to a **CCR5** receptor and (2) an amount of a compound which retards gp41 from adopting a conformation capable of mediating fusion of HIV-1 to a CD4+ cell by binding noncovalently to an epitope on a gp41 fusion intermediate, so as to thereby inhibit HIV-1 infection of the CD4+ cell.

47. A method of inhibiting HIV-1 infection of a CD4+ cell which comprises contacting the CD4+ cell with (1) an amount of an antibody which binds to a **CCR5** receptor, (2) an amount of a compound which retards attachment of HIV-1 to the CD4+ cell by retarding binding of HIV-1 gp120 envelope glycoprotein to CD4 on the surface of the CD4+ cell effective to inhibit HIV-1 infection of the CD4+ cell, and (3) an amount of a compound which retards gp41 from adopting a conformation capable of mediating fusion of HIV-1 to a CD4+ cell by binding noncovalently to an epitope on a gp41 fusion intermediate, so as to thereby inhibit HIV-1 infection of the CD4+ cell.

48. The method of claim 46 or 47, wherein the CD4+ cell is present in a subject and the contacting is effected by administering the compounds to the subject.

49. The method of claim 48, wherein the compounds are administered to the subject simultaneously.

50. The method of claim 48, wherein the compounds are administered to the subject at different times.

51. The method of claim 48, wherein the compounds are administered to the subject by different routes of administration.

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=> d his

     (FILE 'HOME' ENTERED AT 13:34:37 ON 22 JAN 2006)

     FILE 'USPATFULL' ENTERED AT 13:34:46 ON 22 JAN 2006
                E ALLAWAY G P/IN
L1             22 S E4
                E LITWIN V M/IN
L2              9 S E4
L3              1 S L2 NOT L1
                E MADDON P J/IN
L4             47 S E4
L5             33 S L4 NOT (L1 OR L2)
L6             11 S L5 AND (CCR5 OR CC-CKR-5 OR CKR5)

=> s (HIV or human immunodeficiency virus)
         41279 HIV
        483026 HUMAN
         23525 IMMUNODEFICIENCY
         96487 VIRUS
         16786 HUMAN IMMUNODEFICIENCY VIRUS
                  (HUMAN(W)IMMUNODEFICIENCY(W)VIRUS)
L7       43461 (HIV OR HUMAN IMMUNODEFICIENCY VIRUS)

=> s l7 and (CCR5 or CC-CKR-5 or CKR5)
          1930 CCR5
        164045 CC
           589 CKR
       4208072 5
           148 CC-CKR-5
                  (CC(W)CKR(W)5)
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L8 1714 L7 AND (CCR5 OR CC-CKR-5 OR CKR5)

=> s l8 and ay<1997
2436967 AY<1997
L9 10 L8 AND AY<1997

=> s l9 not l1
L10 10 L9 NOT L1

=> d l10,cbib,1-10

L10 ANSWER 1 OF 10 USPATFULL on STN
2002:109167 Mouse CC-**CKR5** receptor polypeptide.
Bergsma, Derk J., Berwyn, PA, United States
Brawner, Mary E., Berwyn, PA, United States
Shabon, Usman, Swarthmore, PA, United States
SmithKline Beecham Corporation, Philadelphia, PA, United States (U.S. corporation)
US 6388055 B1 20020514
APPLICATION: US 1996-724984 19961003 (8)
DOCUMENT TYPE: Utility; GRANTED.
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L10 ANSWER 2 OF 10 USPATFULL on STN
2002:1066 Methods and compositions for polypeptide engineering.
Patten, Phillip A., Mountain View, CA, United States
Stemmer, Willem P. C., Los Gatos, CA, United States
Maxygen, Inc., Redwood City, CA, United States (U.S. corporation)
US 6335160 B1 20020101
APPLICATION: US 1996-769062 19961218 (8)
DOCUMENT TYPE: Utility; GRANTED.
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L10 ANSWER 3 OF 10 USPATFULL on STN
2001:178799 Selenoproteins, coding sequences and methods.
Taylor, Ethan Will, Athens, GA, United States
Nadimpalli, Ram Gopal, Athens, GA, United States
Ramanathan, Chandra Sekar, Athens, GA, United States
University of Georgia Research Foundation, Inc., Athens, GA, United States (U.S. corporation)
US 6303295 B1 20011016
APPLICATION: US 1996-679493 19960712 (8)
PRIORITY: US 1995-1203P 19950714 (60)
US 1995-3112P 19950901 (60)
DOCUMENT TYPE: Utility; GRANTED.
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L10 ANSWER 4 OF 10 USPATFULL on STN
2001:152709 Nucleic acids encoding the G-protein coupled receptor HNFDS78.
Bergsma, Derk, Berwyn, PA, United States
Elshourbagy, Nabil, West Chester, PA, United States
Sarau, Henry, Harleysville, PA, United States
Ruben, Steven, Olney, MD, United States
SmithKline Beecham Corporation, Philadelphia, PA, United States (U.S. corporation)
US 6287801 B1 20010911
APPLICATION: US 1996-681192 19960722 (8)
DOCUMENT TYPE: Utility; GRANTED.
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L10 ANSWER 5 OF 10 USPATFULL on STN
2001:17999 Unique associated Kaposi's Sarcoma virus sequences and uses thereof.
Chang, Yuan, New York, NY, United States
Bohenzky, Roy A., Mountain View, CA, United States
Russo, James J., New York, NY, United States
Edelman, Isidore S., New York, NY, United States
Moore, Patrick S., New York, NY, United States
The Trustees of Columbia University in the City of New York, New York, NY, United States (U.S. corporation)
US 6183751 B1 20010206
APPLICATION: US 1996-757669 19961129 (8)
DOCUMENT TYPE: Utility; Granted.
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L10 ANSWER 6 OF 10 USPATFULL on STN
1999:96489 Methods and compositions for inhibiting **HIV** infection of cells by cleaving **HIV** co-receptor RNA.
Leavitt, Markley C., La Jolla, CA, United States
Tritz, Richard, San Diego, CA, United States
Feng, Yu, San Diego, CA, United States
Barber, Jack, San Diego, CA, United States
Yu, Mang, San Diego, CA, United States

US 5939538 19990817
APPLICATION: US 1996-770235 19961219 (8)
PRIORITY: US 1996-27875P 19961025 (60)
DOCUMENT TYPE: Utility; Granted.
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L10 ANSWER 7 OF 10 USPATFULL on STN
1999:96271 G-coupled receptors associated with macrophage-trophic **HIV**, and diagnostic and therapeutic uses thereof.
Littman, Dan R., New York, NY, United States
Deng, Hongkui, New York, NY, United States
Ellmeier, Wilfried, New York, NY, United States
Landau, Nathaniel R., New York, NY, United States
Liu, Rong, New York, NY, United States
New York University, New York, NY, United States (U.S. corporation)The Aaron Diamond Aids Research Center, New York, NY, United States (U.S. corporation)
US 5939320 19990817
APPLICATION: US 1996-666020 19960619 (8)
PRIORITY: US 1996-17157P 19960520 (60)
DOCUMENT TYPE: Utility; Granted.
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L10 ANSWER 8 OF 10 USPATFULL on STN
1999:89279 Macrophage derived chemokine and chemokine analogs.
Godiska, Ronald, Bothell, WA, United States
Gray, Patrick W., Seattle, WA, United States
ICOS Corporation, Bothell, WA, United States (U.S. corporation)
US 5932703 19990803
APPLICATION: US 1996-660542 19960607 (8)
DOCUMENT TYPE: Utility; Granted.
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L10 ANSWER 9 OF 10 USPATFULL on STN
1998:162655 Kaposi's sarcoma-associated herpesvirus (KSHV) interleukin 6 (IL-6) and uses thereof.
Chang, Yuan, New York, NY, United States
Bohenzky, Roy A., Mountain View, CA, United States
Russo, James J., New York, NY, United States
Edelman, Isidore S., New York, NY, United States
Moore, Patrick S., New York, NY, United States
The Trustees of Columbia University in the City of New York, New York, NY, United States (U.S. corporation)
US 5854398 19981229
APPLICATION: US 1996-748640 19961113 (8)
DOCUMENT TYPE: Utility; Granted.
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L10 ANSWER 10 OF 10 USPATFULL on STN
1998:157173 Polypeptides from Kaposi's sarcoma-associated herpesvirus, DNA encoding same and uses thereof.
Chang, Yuan, New York, NY, United States
Bohenzky, Roy A., Mountian View, CA, United States
Russo, James J., New York, NY, United States
Edelman, Isidore S., New York, NY, United States
Moore, Patrick S., New York, NY, United States
The Trustees of Columbia University in the City of New York, New York, NY, United States (U.S. corporation)
US 5849564 19981215
APPLICATION: US 1996-770379 19961129 (8)
DOCUMENT TYPE: Utility; Granted.
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

=> d his

(FILE 'HOME' ENTERED AT 13:34:37 ON 22 JAN 2006)

FILE 'USPATFULL' ENTERED AT 13:34:46 ON 22 JAN 2006
E ALLAWAY G P/IN
L1 22 S E4
E LITWIN V M/IN
L2 9 S E4
L3 1 S L2 NOT L1
E MADDON P J/IN
L4 47 S E4
L5 33 S L4 NOT (L1 OR L2)
L6 11 S L5 AND (CCR5 OR CC-CKR-5 OR CKR5)
L7 43461 S (HIV OR HUMAN IMMUNODEFICIENCY VIRUS)
L8 1714 S L7 AND (CCR5 OR CC-CKR-5 OR CKR5)
L9 10 S L8 AND AY<1997
L10 10 S L9 NOT L1

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=> file medline
COST IN U.S. DOLLARS                                    SINCE FILE      TOTAL
                                                           ENTRY    SESSION
FULL ESTIMATED COST                                        81.16      81.37

FILE 'MEDLINE' ENTERED AT 13:41:48 ON 22 JAN 2006

 FILE LAST UPDATED: 21 JAN 2006 (20060121/UP).  FILE COVERS 1950 TO DATE.

 On December 11, 2005, the 2006 MeSH terms were loaded.

 The MEDLINE reload for 2006 will soon be available.  For details
 on the 2005 reload, enter HELP RLOAD at an arrow promt (=>).
 See also:

    http://www.nlm.nih.gov/mesh/
    http://www.nlm.nih.gov/pubs/techbull/nd04/nd04_mesh.html
    http://www.nlm.nih.gov/pubs/techbull/nd05/nd05_med_data_changes.html
    http://www.nlm.nih.gov/pubs/techbull/nd05/nd05_2006_MeSH.html

 OLDMEDLINE is covered back to 1950.

 MEDLINE thesauri in the /CN, /CT, and /MN fields incorporate the
 MeSH 2006 vocabulary.

 This file contains CAS Registry Numbers for easy and accurate

=> e allaway g p/au
E1            1     ALLAWAY E C/AU
E2            1     ALLAWAY G/AU
E3           22 --> ALLAWAY G P/AU
E4            3     ALLAWAY GRAHAM/AU
E5            3     ALLAWAY GRAHAM P/AU
E6            3     ALLAWAY J R/AU
E7            1     ALLAWAY JAMES R/AU
E8            3     ALLAWAY M/AU
E9            1     ALLAWAY N/AU
E10           9     ALLAWAY N C/AU
E11           5     ALLAWAY S/AU
E12           8     ALLAWAY S L/AU

=> s e3-e5
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              3 "ALLAWAY GRAHAM"/AU
              3 "ALLAWAY GRAHAM P"/AU
L11          28 ("ALLAWAY G P"/AU OR "ALLAWAY GRAHAM"/AU OR "ALLAWAY GRAHAM
                P"/AU)

=> s l11 and (CCR5 or CC-CKR-5 or CKR5)
           3464 CCR5
          16065 CC
             81 CKR
        2165977 5
              3 CC-CKR-5
                (CC(W)CKR(W)5)
             15 CKR5
L12           4 L11 AND (CCR5 OR CC-CKR-5 OR CKR5)

=> d l12,cbib,1-4

 L12  ANSWER 1 OF 4     MEDLINE on STN
 1998087481.    PubMed ID: 9427609.   AMD3100, a small molecule inhibitor of
      HIV-1 entry via the CXCR4 co-receptor. Donzella G A; Schols D; Lin S W;
      Este J A; Nagashima K A; Maddon P J; Allaway G P; Sakmar T P; Henson G;
      De Clercq E; Moore J P. (The Aaron Diamond AIDS Research Center, The
      Rockefeller University, New York, New York 10016, USA. ) Nature medicine,
      (1998 Jan) 4 (1) 72-7.  Journal code: 9502015. ISSN: 1078-8956. Pub.
      country: United States. Language: English.

 L12  ANSWER 2 OF 4     MEDLINE on STN
 1998080414.    PubMed ID: 9420225.   Amino-terminal substitutions in the CCR5
      coreceptor impair gp120 binding and human immunodeficiency virus type 1
      entry. Dragic T; Trkola A; Lin S W; Nagashima K A; Kajumo F; Zhao L; Olson
      W C; Wu L; Mackay C R; Allaway G P; Sakmar T P; Moore J P; Maddon P J.
      (Aaron Diamond AIDS Research Center, The Rockefeller University, New York,
      New York 10016, USA.. tdragic@adarc.org) . Journal of virology, (1998 Jan)
      72 (1) 279-85.  Journal code: 0113724. ISSN: 0022-538X. Pub. country:
      United States. Language: English.

 L12  ANSWER 3 OF 4     MEDLINE on STN
 97064177.    PubMed ID: 8906796.   CD4-dependent, antibody-sensitive
      interactions between HIV-1 and its co-receptor CCR-5. Trkola A; Dragic T;
```

Maddon P J; Moore J P. (The Aaron Diamond AIDS Research Centre, The Rockefeller University, New York 10016, USA.) Nature, (1996 Nov 14) 384 (6605) 184-7. Journal code: 0410462. ISSN: 0028-0836. Pub. country: ENGLAND: United Kingdom. Language: English.

L12 ANSWER 4 OF 4 MEDLINE on STN
96260018. PubMed ID: 8649512. HIV-1 entry into CD4+ cells is mediated by the chemokine receptor **CC-CKR-5**. Dragic T; Litwin V; **Allaway G P**; Martin S R; Huang Y; Nagashima K A; Cayanan C; Maddon P J; Koup R A; Moore J P; Paxton W A. (The Aaron Diamond AIDS Research Center, The Rockefeller University, New York 10016, USA.) Nature, (1996 Jun 20) 381 (6584) 667-73. Journal code: 0410462. ISSN: 0028-0836. Pub. country: ENGLAND: United Kingdom. Language: English.

=> d l12,cbib,ab,1-4

L12 ANSWER 1 OF 4 MEDLINE on STN
1998087481. PubMed ID: 9427609. AMD3100, a small molecule inhibitor of HIV-1 entry via the CXCR4 co-receptor. Donzella G A; Schols D; Lin S W; Este J A; Nagashima K A; Maddon P J; **Allaway G P**; Sakmar T P; Henson G; De Clercq E; Moore J P. (The Aaron Diamond AIDS Research Center, The Rockefeller University, New York, New York 10016, USA.) Nature medicine, (1998 Jan) 4 (1) 72-7. Journal code: 9502015. ISSN: 1078-8956. Pub. country: United States. Language: English.
AB The bicyclam AMD3100 (formula weight 830) blocks HIV-1 entry and membrane fusion via the CXCR4 co-receptor, but not via **CCR5**. AMD3100 prevents monoclonal antibody 12G5 from binding to CXCR4, but has no effect on binding of monoclonal antibody 2D7 to **CCR5**. It also inhibits binding of the CXC-chemokine, SDF-1alpha, to CXCR4 and subsequent signal transduction, but does not itself cause signaling and has no effect on RANTES signaling via **CCR5**. Thus, AMD3100 prevents CXCR4 functioning as both a HIV-1 co-receptor and a CXC-chemokine receptor. Development of small molecule inhibitors of HIV-1 entry is feasible.

L12 ANSWER 2 OF 4 MEDLINE on STN
1998080414. PubMed ID: 9420225. Amino-terminal substitutions in the **CCR5** coreceptor impair gp120 binding and human immunodeficiency virus type 1 entry. Dragic T; Trkola A; Lin S W; Nagashima K A; Kajumo F; Zhao L; Olson W C; Wu L; Mackay C R; **Allaway G P**; Sakmar T P; Moore J P; Maddon P J. (Aaron Diamond AIDS Research Center, The Rockefeller University, New York, New York 10016, USA.. tdragic@adarc.org) . Journal of virology, (1998 Jan) 72 (1) 279-85. Journal code: 0113724. ISSN: 0022-538X. Pub. country: United States. Language: English.
AB The CC-chemokine receptor **CCR5** is required for the efficient fusion of macrophage (M)-tropic human immunodeficiency virus type 1 (HIV-1) strains with the plasma membrane of CD4+ cells and interacts directly with the viral surface glycoprotein gp120. Although receptor chimera studies have provided useful information, the domains of **CCR5** that function for HIV-1 entry, including the site of gp120 interaction, have not been unambiguously identified. Here, we use site-directed, alanine-scanning mutagenesis of **CCR5** to show that substitutions of the negatively charged aspartic acid residues at positions 2 and 11 (D2A and D11A) and a glutamic acid residue at position 18 (E18A), individually or in combination, impair or abolish **CCR5**-mediated HIV-1 entry for the ADA and JR-FL M-tropic strains and the DH123 dual-tropic strain. These mutations also impair Env-mediated membrane fusion and the gp120-**CCR5** interaction. Of these three residues, only D11 is necessary for CC-chemokine-mediated inhibition of HIV-1 entry, which is, however, also dependent on other extracellular **CCR5** residues. Thus, the gp120 and CC-chemokine binding sites on **CCR5** are only partially overlapping, and the former site requires negatively charged residues in the amino-terminal **CCR5** domain.

L12 ANSWER 3 OF 4 MEDLINE on STN
97064177. PubMed ID: 8906796. CD4-dependent, antibody-sensitive interactions between HIV-1 and its co-receptor CCR-5. Trkola A; Dragic T; Arthos J; Binley J M; Olson W C; **Allaway G P**; Cheng-Mayer C; Robinson J; Maddon P J; Moore J P. (The Aaron Diamond AIDS Research Centre, The Rockefeller University, New York 10016, USA.) Nature, (1996 Nov 14) 384 (6605) 184-7. Journal code: 0410462. ISSN: 0028-0836. Pub. country: ENGLAND: United Kingdom. Language: English.
AB The beta-chemokine receptor CCR-5 is an essential co-factor for fusion of HIV-1 strains of the non-syncytium-inducing (NSI) phenotype with CD4+ T-cells. The primary binding site for human immunodeficiency virus (HIV)-1 is the CD4 molecule, and the interaction is mediated by the viral surface glycoprotein gp120 (refs 6, 7). The mechanism of CCR-5 function during HIV-1 entry has not been defined, but we have shown previously that its beta-chemokine ligands prevent HIV-1 from fusing with the cell. We therefore investigated whether CCR-5 acts as a second binding site for HIV-1 simultaneously with or subsequent to the interaction between gp120 and CD4. We used a competition assay based on gp120 inhibition of the binding of the CCR-5 ligand, macrophage inflammatory protein (MIP)-1beta,

We conclude that CD4 binding, although not absolutely necessary for the gp120-CCR-5 interaction, greatly increases its efficiency. Neutralizing monoclonal antibodies against several sites on gp120, including the V3 loop and CD4-induced epitopes, inhibited the interaction of gp120 with CCR-5, without affecting gp120-CD4 binding. Interference with HIV-1 binding to one or both of its receptors (CD4 and CCR-5) may be an important mechanism of virus neutralization.

L12 ANSWER 4 OF 4 MEDLINE on STN
96260018. PubMed ID: 8649512. HIV-1 entry into CD4+ cells is mediated by the chemokine receptor **CC-CKR-5**. Dragic T; Litwin V; **Allaway G P**; Martin S R; Huang Y; Nagashima K A; Cayanan C; Maddon P J; Koup R A; Moore J P; Paxton W A. (The Aaron Diamond AIDS Research Center, The Rockefeller University, New York 10016, USA.) Nature, (1996 Jun 20) 381 (6584) 667-73. Journal code: 0410462. ISSN: 0028-0836. Pub. country: ENGLAND: United Kingdom. Language: English.
AB The beta-chemokines MIP-1alpha, MIP-1beta and RANTES inhibit infection of CD4+ T cells by primary, non-syncytium-inducing (NSI) HIV-1 strains at the virus entry stage, and also block env-mediated cell-cell membrane fusion. CD4+ T cells from some HIV-1-exposed uninfected individuals cannot fuse with NSI HIV-1 strains and secrete high levels of beta-chemokines. Expression of the beta-chemokine receptor **CC-CKR-5** in CD4+, non-permissive human and non-human cells renders them susceptible to infection by NSI strains, and allows env-mediated membrane fusion. **CC-CKR-5** is a second receptor for NSI primary viruses.

```
=> e litwin v m/au
E1          10     LITWIN V/AU
E2           1     LITWIN V A/AU
E3           0 --> LITWIN V M/AU
E4           2     LITWIN W/AU
E5           1     LITWIN W J/AU
E6           1     LITWIN Y/AU
E7           1     LITWINCZAK ALTSCHULER S/AU
E8           6     LITWINCZUK R A/AU
E9           2     LITWINENKO A/AU
E10          1     LITWINENKO J W/AU
E11          8     LITWINIENKO GRZEGORZ/AU
E12          1     LITWINIENKO J/AU

=> e maddon p j/au
E1           3     MADDON D E/AU
E2           1     MADDON P/AU
E3          36 --> MADDON P J/AU
E4           6     MADDON PAUL J/AU
E5           1     MADDONNI GABRIELA/AU
E6           1     MADDONNI GUSTAVO ANGEL/AU
E7           2     MADDOUX G/AU
E8           7     MADDOUX G L/AU
E9           1     MADDOUX L D/AU
E10          1     MADDOW CHARLES/AU
E11          3     MADDOW CHARLES L/AU
E12          1     MADDOW J/AU

=> s e2-e4
             1 "MADDON P"/AU
            36 "MADDON P J"/AU
             6 "MADDON PAUL J"/AU
L13         43 ("MADDON P"/AU OR "MADDON P J"/AU OR "MADDON PAUL J"/AU)

=> d his

     (FILE 'HOME' ENTERED AT 13:34:37 ON 22 JAN 2006)

     FILE 'USPATFULL' ENTERED AT 13:34:46 ON 22 JAN 2006
                E ALLAWAY G P/IN
L1           22 S E4
                E LITWIN V M/IN
L2            9 S E4
L3            1 S L2 NOT L1
                E MADDON P J/IN
L4           47 S E4
L5           33 S L4 NOT (L1 OR L2)
L6           11 S L5 AND (CCR5 OR CC-CKR-5 OR CKR5)
L7        43461 S (HIV OR HUMAN IMMUNODEFICIENCY VIRUS)
L8         1714 S L7 AND (CCR5 OR CC-CKR-5 OR CKR5)
L9           10 S L8 AND AY<1997
L10          10 S L9 NOT L1

     FILE 'MEDLINE' ENTERED AT 13:41:48 ON 22 JAN 2006
                E ALLAWAY G P/AU
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L12           4 S L11 AND (CCR5 OR CC-CKR-5 OR CKR5)
                E LITWIN V M/AU
                E MADDON P J/AU
L13          43 S E2-E4

=> s l13 not l11
L14          33 L13 NOT L11

=> s l14 and (CCR5 or CC-CKR-5 or CKR5)
          3464 CCR5
         16065 CC
            81 CKR
       2165977 5
             3 CC-CKR-5
                 (CC(W)CKR(W)5)
            15 CKR5
L15          3 L14 AND (CCR5 OR CC-CKR-5 OR CKR5)

=> d l15,cbib,1-3

L15  ANSWER 1 OF 3     MEDLINE on STN
2002376955.     PubMed ID: 12097589.   Oligomeric and conformational properties
     of a proteolytically mature, disulfide-stabilized human immunodeficiency
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     Sanders Rogier W; Zhu Ping; Lu Min; Anselma Deborah J; Villa Anthony R;
     Parren Paul W H I; Binley James M; Roux Kenneth H; Maddon Paul J; Moore
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     York 10591, USA. ) Journal of virology, (2002 Aug) 76 (15) 7760-76.
     Journal code: 0113724. ISSN: 0022-538X. Pub. country: United States.
     Language: English.

L15  ANSWER 2 OF 3     MEDLINE on STN
2001092646.     PubMed ID: 11134270.   Potent, broad-spectrum inhibition of
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     Journal code: 0113724. ISSN: 0022-538X. Pub. country: United States.
     Language: English.

L15  ANSWER 3 OF 3     MEDLINE on STN
1999214354.     PubMed ID: 10196311.   Differential inhibition of human
     immunodeficiency virus type 1 fusion, gp120 binding, and CC-chemokine
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     Nagashima K A; Tran D N; Anselma D J; Monard S P; Segal J P; Thompson D A;
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     virology, (1999 May) 73 (5) 4145-55.  Journal code: 0113724. ISSN:
     0022-538X. Pub. country: United States. Language: English.


=> d his

     (FILE 'HOME' ENTERED AT 13:34:37 ON 22 JAN 2006)

     FILE 'USPATFULL' ENTERED AT 13:34:46 ON 22 JAN 2006
                E ALLAWAY G P/IN
L1           22 S E4
                E LITWIN V M/IN
L2            9 S E4
L3            1 S L2 NOT L1
                E MADDON P J/IN
L4           47 S E4
L5           33 S L4 NOT (L1 OR L2)
L6           11 S L5 AND (CCR5 OR CC-CKR-5 OR CKR5)
L7        43461 S (HIV OR HUMAN IMMUNODEFICIENCY VIRUS)
L8         1714 S L7 AND (CCR5 OR CC-CKR-5 OR CKR5)
L9           10 S L8 AND AY<1997
L10          10 S L9 NOT L1

     FILE 'MEDLINE' ENTERED AT 13:41:48 ON 22 JAN 2006
                E ALLAWAY G P/AU
L11          28 S E3-E5
L12           4 S L11 AND (CCR5 OR CC-CKR-5 OR CKR5)
                E LITWIN V M/AU
                E MADDON P J/AU
L13          43 S E2-E4
L14          33 S L13 NOT L11
L15           3 S L14 AND (CCR5 OR CC-CKR-5 OR CKR5)

=> s (HIV or human immunodeficiency virus)
        155134 HIV
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        121217 IMMUNODEFICIENCY
        406226 VIRUS
         47625 HUMAN IMMUNODEFICIENCY VIRUS
                 (HUMAN(W)IMMUNODEFICIENCY(W)VIRUS)
L16     160375 (HIV OR HUMAN IMMUNODEFICIENCY VIRUS)

=> s l16 and (CCR5 or CC-CKR-5 or CKR5)
          3464 CCR5
         16065 CC
            81 CKR
       2165977 5
             3 CC-CKR-5
                 (CC(W)CKR(W)5)
            15 CKR5
L17        2456 L16 AND (CCR5 OR CC-CKR-5 OR CKR5)

=> s l17 and py<1997
      11054681 PY<1997
L18          36 L17 AND PY<1997

=> d l18,cbib,1-36

L18  ANSWER 1 OF 36     MEDLINE on STN
2001280510.    PubMed ID: 11363902.   Basic science discovery: opening the door
     on HIV. Anonymous. PI perspective, (1996 Sep) (No 19) 17-8. Journal
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     English.

L18  ANSWER 2 OF 36     MEDLINE on STN
2001280414.    PubMed ID: 11363806.   NIAID researchers identify cofactors for
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     country: United States. Language: English.

L18  ANSWER 3 OF 36     MEDLINE on STN
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     States. Language: English.

L18  ANSWER 4 OF 36     MEDLINE on STN
97136461.    PubMed ID: 8984652.   New hope in HIV disease. Balter M.
     Science, (1996 Dec 20) 274 (5295) 1988-9. Journal code: 0404511. ISSN:
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L18  ANSWER 5 OF 36     MEDLINE on STN
97130437.    PubMed ID: 8976200.   Efficient interaction of HIV-1 with
     purified dendritic cells via multiple chemokine coreceptors.
     Granelli-Piperno A; Moser B; Pope M; Chen D; Wei Y; Isdell F; O'Doherty U;
     Paxton W; Koup R; Mojsov S; Bhardwaj N; Clark-Lewis I; Baggiolini M;
     Steinman R M. (Theodor Kocher Institute, Bern, Switzerland. ) Journal of
     experimental medicine, (1996 Dec 1) 184 (6) 2433-8. Journal code:
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L18  ANSWER 6 OF 36     MEDLINE on STN
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L18  ANSWER 7 OF 36     MEDLINE on STN
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     virology, (1996 Dec) 70 (12) 8355-60. Journal code: 0113724. ISSN:
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L18  ANSWER 8 OF 36     MEDLINE on STN
97125644.    PubMed ID: 8970716.   Summary of track A: basic science. Birx D L;
     VanCott T; Michael N; McNeil J; Stamatos N; Gilliam B; Davis R; Carr J;
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     Research, Rockville, Maryland, USA. ) AIDS (London, England), (1996 Dec)
     10 Suppl 3 S85-106. Ref: 150. Journal code: 8710219. ISSN: 0269-9370.
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L18  ANSWER 9 OF 36     MEDLINE on STN
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University of California, San Francisco, Post Office Box 419100, San Francisco, CA 94141-9100, USA.. mark_goldsmith@quickmail.ucsf.edu) . Science, **(1996 Dec 13)** 274 (5294) 1924-6. Journal code: 0404511. ISSN: 0036-8075. Pub. country: United States. Language: English.

L18 ANSWER 10 OF 36 MEDLINE on STN
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L18 ANSWER 11 OF 36 MEDLINE on STN
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L18 ANSWER 12 OF 36 MEDLINE on STN
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L18 ANSWER 13 OF 36 MEDLINE on STN
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L18 ANSWER 14 OF 36 MEDLINE on STN
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L18 ANSWER 15 OF 36 MEDLINE on STN
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L18 ANSWER 16 OF 36 MEDLINE on STN
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L18 ANSWER 18 OF 36 MEDLINE on STN
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Language: English.

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L18 ANSWER 23 OF 36 MEDLINE on STN
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L18 ANSWER 24 OF 36 MEDLINE on STN
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L18 ANSWER 25 OF 36 MEDLINE on STN
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L18 ANSWER 27 OF 36 MEDLINE on STN
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L18 ANSWER 30 OF 36 MEDLINE on STN
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L18 ANSWER 31 OF 36 MEDLINE on STN
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L18 ANSWER 32 OF 36 MEDLINE on STN
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L18 ANSWER 33 OF 36 MEDLINE on STN
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L18 ANSWER 34 OF 36 MEDLINE on STN
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L18 ANSWER 35 OF 36 MEDLINE on STN
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L18 ANSWER 36 OF 36 MEDLINE on STN
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=> d l18,cbib,ab,1-36

L18 ANSWER 1 OF 36 MEDLINE on STN
2001280510. PubMed ID: 11363902. Basic science discovery: opening the door on **HIV**. Anonymous. PI perspective, **(1996 Sep)** (No 19) 17-8. Journal code: 9102818. ISSN: 1058-7454. Pub. country: United States. Language: English.
AB Back-to-back discoveries of chemokines and the **CKR5** receptor site shed new light on how **HIV** infects cells and why there is so much difference in individual response. The roles of **CKR5** and fusin in **HIV** disease development are discussed. How defects in the **CKR5** receptor protein may give these people some immunity to **HIV** infection is explained. The clinical implications involved in studying this process are highlighted.

L18 ANSWER 2 OF 36 MEDLINE on STN
2001280414. PubMed ID: 11363806. NIAID researchers identify cofactors for entry of **HIV** into cells. National Institute of Allergy and Infectious Diseases. Folkers G. NIAID AIDS agenda / National Institute of Allergy and Infectious Diseases, **(1996 Jun)** 1, 10-1. Journal code: 9432911. Pub. country: United States. Language: English.
AB Scientists at the National Institute of Allergy and Infectious Diseases (NIAID) report that they have identified fusion cofactors that, for certain strains of **HIV**-1, make both human and nonhuman CD4+ cells susceptible to **HIV** fusion and infection. Researchers demonstrated that these **HIV**-1 strains require, in addition to CD4, a cell surface molecule called CC **CKR5** in order to fuse with the membranes of immune system cells. The NIAID researchers found that macrophage-tropic isolates (the strains found in patients during the symptom-free stage of **HIV** disease) failed to fuse with cells expressing only CD4, but fused with cells that have both CD4 and CC **CKR5**. Researchers also learned that CC **CKR5** is a

HIV infection in cells. The data suggest that one way these chemokines suppress **HIV** infectivity is by blocking the fusion process used by the virus to enter cells. NIAID also learned earlier this year of another fusion cofactor, termed fusin, which is used by other strains of **HIV** for entry into immune cells. The findings help provide insights into the pathogenesis of **HIV** disease and suggest new approaches to developing animal models of **HIV** infection.

L18 ANSWER 3 OF 36 MEDLINE on STN
97138999. PubMed ID: 8985971. Scientists confirm natural resistance to **HIV**-1. Anonymous. Oncology (Williston Park, N.Y.), **(1996 Dec)** 10 (12) 1879-80. Journal code: 8712059. ISSN: 0890-9091. Pub. country: United States. Language: English.

L18 ANSWER 4 OF 36 MEDLINE on STN
97136461. PubMed ID: 8984652. New hope in **HIV** disease. Balter M. Science, **(1996 Dec 20)** 274 (5295) 1988-9. Journal code: 0404511. ISSN: 0036-8075. Pub. country: United States. Language: English.

L18 ANSWER 5 OF 36 MEDLINE on STN
97130437. PubMed ID: 8976200. Efficient interaction of **HIV**-1 with purified dendritic cells via multiple chemokine coreceptors. Granelli-Piperno A; Moser B; Pope M; Chen D; Wei Y; Isdell F; O'Doherty U; Paxton W; Koup R; Mojsov S; Bhardwaj N; Clark-Lewis I; Baggiolini M; Steinman R M. (Theodor Kocher Institute, Bern, Switzerland.) Journal of experimental medicine, **(1996 Dec 1)** 184 (6) 2433-8. Journal code: 2985109R. ISSN: 0022-1007. Pub. country: United States. Language: English.
AB **HIV**-1 actively replicates in dendritic cell (DC)-T cell cocultures, but it has been difficult to demonstrate substantial infection of purified mature DCs. We now find that **HIV**-1 begins reverse transcription much more efficiently in DCs than T cells, even though T cells have higher levels of CD4 and gp120 binding. DCs isolated from skin or from blood precursors behave similarly. Several M-tropic strains and the T-tropic strain IIIB enter DCs efficiently, as assessed by the progressive formation of the early products of reverse transcription after a 90-min virus pulse at 37 degrees C. However, few late gag-containing sequences are detected, so that active viral replication does not occur. The formation of these early transcripts seems to follow entry of **HIV**-1, rather than binding of virions that contain viral DNA. Early transcripts are scarce if DCs are exposed to virus on ice for 4 h, or for 90 min at 37 degrees C, conditions which allow virus binding. Also the early transcripts once formed are insensitive to trypsin. The entry of a M-tropic isolates is blocked by the chemokine RANTES, and the entry of IIIB by SDF-1. RANTES interacts with **CCR5** and SDF-1 with CXCR4 receptors. Entry of M-tropic but not T-tropic virus is ablated in DCs from individuals who lack a functional **CCR5** receptor. DCs express more **CCR5** and CXCR4 mRNA than T cells. Therefore, while **HIV**-1 does not replicate efficiently in mature DCs, viral entry can be active and can be blocked by chemokines that act on known receptors for M- and T-tropic virus.

L18 ANSWER 6 OF 36 MEDLINE on STN
97129941. PubMed ID: 8984621. Ethics of AIDS study. Gomperts E D; Donfield S M. Science, **(1996 Dec 6)** 274 (5293) 1596. Journal code: 0404511. ISSN: 0036-8075. Pub. country: United States. Language: English.

L18 ANSWER 7 OF 36 MEDLINE on STN
97126031. PubMed ID: 8970955. Primary, syncytium-inducing **human immunodeficiency virus** type 1 isolates are dual-tropic and most can use either Lestr or **CCR5** as coreceptors for virus entry. Simmons G; Wilkinson D; Reeves J D; Dittmar M T; Beddows S; Weber J; Carnegie G; Desselberger U; Gray P W; Weiss R A; Clapham P R. (Virology Laboratory, The Institute of Cancer Research, London, United Kingdom.) Journal of virology, **(1996 Dec)** 70 (12) 8355-60. Journal code: 0113724. ISSN: 0022-538X. Pub. country: United States. Language: English.
AB A panel of primary syncytium-inducing (SI) **human immunodeficiency virus** type 1 isolates that infected several CD4+ T-cell lines, including MT-2 and C8166, were tested for infection of blood-derived macrophages. Infectivity titers for C8166 cells and macrophages demonstrated that primary SI strains infected macrophages much more efficiently than T-cell line-adapted **HIV**-1 strains such as LAI and RF. These primary SI strains were therefore dual-tropic. Nine biological clones of two SI strains, prepared by limiting dilution, had macrophage/C8166 infectivity ratios similar to those of their parental viruses, indicating that the dual-tropic phenotype was not due to a mixture of non-SI/macrophage-tropic and SI/T-cell tropic viruses. We tested whether the primary SI strains used either Lestr (fusin) or **CCR5** as coreceptors. Infection of cat CCC/CD4 cells transiently expressing Lestr supported infection by T-cell line-adapted strains including LAI, whereas CCC/CD4 cells expressing **CCR5** were sensitive to primary non-SI strains as well as to the molecularly cloned strains SF-162 and JR-CSF. Several primary SI strains, as well as the molecularly cloned dual-tropic viruses 89.6 and GUN-1,

choose between Lestr and **CCR5** for entry into cells. Interestingly, some dual-tropic primary SI strains that infected Lestr+ cells failed to infect **CCR5**+ cells, suggesting that these viruses may use an alternative coreceptor for infection of macrophages. Alternatively, **CCR5** may be processed or presented differently on cat cells so that entry of some primary SI strains but not others is affected.

L18 ANSWER 8 OF 36 MEDLINE on STN

97125644. PubMed ID: 8970716. Summary of track A: basic science. Birx D L; VanCott T; Michael N; McNeil J; Stamatos N; Gilliam B; Davis R; Carr J; McCutchan F. (Division of Retrovirology, Walter Reed Army Institute of Research, Rockville, Maryland, USA.) AIDS (London, England), **(1996 Dec)** 10 Suppl 3 S85-106. Ref: 150. Journal code: 8710219. ISSN: 0269-9370. Pub. country: United States. Language: English.

AB AIM: To review Track A, which is organized into five broad areas of emphasis. TOOLS: A variety of new virologic tools are allowing researchers to more effectively evaluate many aspects of **HIV**, from various therapies and vaccine candidates to the recombination and international spread of genotypes. PATHOGENESIS: The recent understandings of **HIV**-1 pathogenesis have led to potential new treatment strategies of early aggressive treatment with combination drugs and the potential for biologic or immunologic therapy directed to blocking viral entry through second receptors. TREATMENT: **HIV** treatment focused on chemical/drug advances and treatment, and immunologic/genetic advances. Some areas of development include optimizing combination therapies using the oncology model; continued work on new preclinical compounds (e.g. integrase and tat/rev inhibitors); evaluation of viral reduction in all compartments; and resistance surveillance and prevention. Biologics, including fusin/CC-**CKR5** inhibitors and CD8 **HIV**-1 suppressor factors, ex vivo expansion of T cells and in vivo expansion of effector CD8 cells continue to be developed as possible future treatments. VACCINES: In order to obtain worldwide control over **HIV**, we must have a universally effective vaccine. The question remains as to what specifically is required for a protective response. Mechanisms of CD8 suppression, and cellular and antibody correlates of protection were discussed as areas of research that may shed light on the critical protective immune response. GENOTYPES: Discussion of **HIV** genotypes focused on international subtypes, correlates of diversity, and **HIV**-1 recombination. Numerous groups have shown an international intermixing of **HIV**-1 strains. Recombination during transcription was found to lead to extensive genomic shift and increased diversity, which may also increase **HIV**-1 fitness and enhance transmission. CONCLUSIONS: The spread and adaptation of **HIV**-1 is occurring independently of borders. Therefore, **HIV**-1 research must be global; vaccine development must be international in concept and application; collaboration in all areas is essential for success in combating **HIV**; and finally, the challenge for the future will be to actively involve all basic scientists in the science of the international epidemics.

L18 ANSWER 9 OF 36 MEDLINE on STN

97102599. PubMed ID: 8943208. Multiple extracellular elements of **CCR5** and **HIV**-1 entry: dissociation from response to chemokines. Atchison R E; Gosling J; Monteclaro F S; Franci C; Digilio L; Charo I F; Goldsmith M A. (Gladstone Institute of Virology and Immunology, School of Medicine, University of California, San Francisco, Post Office Box 419100, San Francisco, CA 94141-9100, USA.. mark_goldsmith@quickmail.ucsf.edu) . Science, **(1996 Dec 13)** 274 (5294) 1924-6. Journal code: 0404511. ISSN: 0036-8075. Pub. country: United States. Language: English.

AB The human beta-chemokine receptor **CCR5** is an important cofactor for entry of **human immunodeficiency virus**-type 1 (**HIV**-1). The murine form of **CCR5**, despite its 82 percent identity to the human form, was not functional as an **HIV**-1 coreceptor. **HIV**-1 entry function could be reconstituted by fusion of various individual elements derived from the extracellular region of human **CCR5** onto murine **CCR5**. Analysis of chimeras containing elements from human **CCR5** and human CCR2B suggested that a complex structure rather than single contact sites is responsible for facilitation of viral entry. Further, certain chimeras lacking the domains necessary to signal in response to their natural chemokine ligands retained vigorous **HIV**-1 coreceptor activity.

L18 ANSWER 10 OF 36 MEDLINE on STN

97094384. PubMed ID: 8939871. Molecular mimicry of human cytokine and cytokine response pathway genes by KSHV. Moore P S; Boshoff C; Weiss R A; Chang Y. (School of Public Health, Columbia University, New York, NY 10032, USA.) Science, **(1996 Dec 6)** 274 (5293) 1739-44. Journal code: 0404511. ISSN: 0036-8075. Pub. country: United States. Language: English.

AB Four virus proteins similar to two human macrophage inflammatory protein (MIP) chemokines, interleukin-6 (IL-6), and interferon regulatory factor (IRF) are encoded by the Kaposi's sarcoma-associated herpesvirus (KSHV) genome. vIL-6 was functional in B9 proliferation assays and primarily expressed in KSHV-infected hematopoietic cells rather than KS lesions. **HIV**-1 transmission studies showed that vMIP-I is similar to human MIP

dependent on the **CCR5** co-receptor. These viral genes may form part of the response to host defenses contributing to virus-induced neoplasia and may have relevance to KSHV and **HIV**-I interactions.

L18 ANSWER 11 OF 36 MEDLINE on STN
97066810. PubMed ID: 8928003. Investigators detail **HIV**'s fatal handshake. Cohen J. Science, **(1996 Oct 25)** 274 (5287) 502. Journal code: 0404511. ISSN: 0036-8075. Pub. country: United States. Language: English.

L18 ANSWER 12 OF 36 MEDLINE on STN
97064177. PubMed ID: 8906796. CD4-dependent, antibody-sensitive interactions between **HIV**-1 and its co-receptor CCR-5. Trkola A; Dragic T; Arthos J; Binley J M; Olson W C; Allaway G P; Cheng-Mayer C; Robinson J; Maddon P J; Moore J P. (The Aaron Diamond AIDS Research Centre, The Rockefeller University, New York 10016, USA.) Nature, **(1996 Nov 14)** 384 (6605) 184-7. Journal code: 0410462. ISSN: 0028-0836. Pub. country: ENGLAND: United Kingdom. Language: English.
AB The beta-chemokine receptor CCR-5 is an essential co-factor for fusion of **HIV**-1 strains of the non-syncytium-inducing (NSI) phenotype with CD4+ T-cells. The primary binding site for **human immunodeficiency virus** (**HIV**)-1 is the CD4 molecule, and the interaction is mediated by the viral surface glycoprotein gp120 (refs 6, 7). The mechanism of CCR-5 function during **HIV**-1 entry has not been defined, but we have shown previously that its beta-chemokine ligands prevent **HIV**-1 from fusing with the cell. We therefore investigated whether CCR-5 acts as a second binding site for **HIV**-1 simultaneously with or subsequent to the interaction between gp120 and CD4. We used a competition assay based on gp120 inhibition of the binding of the CCR-5 ligand, macrophage inflammatory protein (MIP)-1beta, to its receptor on activated CD4+ T cells or CCR-5-positive CD4- cells. We conclude that CD4 binding, although not absolutely necessary for the gp120-CCR-5 interaction, greatly increases its efficiency. Neutralizing monoclonal antibodies against several sites on gp120, including the V3 loop and CD4-induced epitopes, inhibited the interaction of gp120 with CCR-5, without affecting gp120-CD4 binding. Interference with **HIV**-1 binding to one or both of its receptors (CD4 and CCR-5) may be an important mechanism of virus neutralization.

L18 ANSWER 13 OF 36 MEDLINE on STN
97064176. PubMed ID: 8906795. CD4-induced interaction of primary **HIV**-1 gp120 glycoproteins with the chemokine receptor CCR-5. Wu L; Gerard N P; Wyatt R; Choe H; Parolin C; Ruffing N; Borsetti A; Cardoso A A; Desjardin E; Newman W; Gerard C; Sodroski J. (LeukoSite, Inc., Cambridge, Massachusetts 02142, USA.) Nature, **(1996 Nov 14)** 384 (6605) 179-83. Journal code: 0410462. ISSN: 0028-0836. Pub. country: ENGLAND: United Kingdom. Language: English.
AB For efficient entry into target cells, primary macrophage-tropic and laboratory-adapted human immunodeficiency viruses type 1 (**HIV**-1) require particular chemokine receptors, CCR-5 and CXCR-4, respectively, as well as the primary receptor CD4 (refs 1-6). Here we show that a complex of gp120, the exterior envelope glycoprotein, of macrophage-tropic primary **HIV**-1 and soluble CD4 interacts specifically with CCR-5 and inhibits the binding of the natural CCR-5 ligands, macrophage inflammatory protein (MIP)-1alpha and MIP-1beta (refs 7, 8). The apparent affinity of the interaction between gp120 and CCR-5 was dramatically lower in the absence of soluble CD4. Additionally, in the absence of gp120, an interaction between a two-domain CD4 fragment and CCR-5 was observed. A gp120 fragment retaining the CD4-binding site and overlapping epitopes was able to interact with CCR-5 only if the V3 loop, which can specify **HIV**-1 tropism and chemokine receptor choice, was also present on the molecule. Neutralizing antibodies directed against either CD4-induced or V3 epitopes on gp120 blocked the interaction of gp120-CD4 complexes with CCR-5. These results suggest that **HIV**-1 attachment to CD4 creates a high-affinity binding site for CCR-5, leading to membrane fusion and virus entry.

L18 ANSWER 14 OF 36 MEDLINE on STN
97064163. PubMed ID: 8906782. **HIV**. One on one meets two. Wain-Hobson S. Nature, **(1996 Nov 14)** 384 (6605) 117-8. Journal code: 0410462. ISSN: 0028-0836. Pub. country: ENGLAND: United Kingdom. Language: English.

L18 ANSWER 15 OF 36 MEDLINE on STN
97054456. PubMed ID: 8898753. The V3 domain of the **HIV**-1 gp120 envelope glycoprotein is critical for chemokine-mediated blockade of infection. Cocchi F; DeVico A L; Garzino-Demo A; Cara A; Gallo R C; Lusso P. (Institute of Human Virology, University of Maryland Biotechnology Institute & School of Medicine, Baltimore, Maryland 21201, USA.) Nature medicine, **(1996 Nov)** 2 (11) 1244-7. Journal code: 9502015. ISSN: 1078-8956. Pub. country: United States. Language: English.
AB The ability of CD8 T cells derived from **human immunodeficiency virus** (**HIV**)-infected patients to produce soluble **HIV**-suppressive factor(s) (**HIV**-SF) has been suggested as an important mechanism of control of

beta were recently identified as the major components of the **HIV**-SF produced by both immortalized and primary patient CD8 T cells. Whereas they potently inhibit infection by primary and macrophage-tropic **HIV**-1 isolates, T-cell line-adapted viral strains tend to be insensitive to their suppressive effects. Consistent with this discrepancy, two distinct chemokine receptors, namely, CXCR4 (ref. 7) and **CCR5** (ref. 8), were recently identified as potential co-receptors for T-cell line-adapted and macrophage-tropic **HIV**-1 isolates, respectively. Here, we demonstrate that the third hypervariable domain of the gp 120 envelope glycoprotein is a critical determinant of the susceptibility of **HIV**-1 to chemokines. Moreover, we show that RANTES, MIP-1 alpha and MIP-1 beta block the entry of **HIV**-1 into cells and that their antiviral activity is independent of pertussis toxin-sensitive signal transduction pathways mediated by chemokine receptors. The ability of the chemokines to block the early steps of **HIV** infection could be exploited to develop novel therapeutic approaches for AIDS.

L18 ANSWER 16 OF 36 MEDLINE on STN
97054455. PubMed ID: 8898752. The role of a mutant **CCR5** allele in **HIV**-1 transmission and disease progression. Huang Y; Paxton W A; Wolinsky S M; Neumann A U; Zhang L; He T; Kang S; Ceradini D; Jin Z; Yazdanbakhsh K; Kunstman K; Erickson D; Dragon E; Landau N R; Phair J; Ho D D; Koup R A. (Aaron Diamond AIDS Research Center, New York, New York, USA.) Nature medicine, **(1996 Nov)** 2 (11) 1240-3. Journal code: 9502015. ISSN: 1078-8956. Pub. country: United States. Language: English.
AB A 32-nucleotide deletion (delta 32) within the beta-chemokine receptor 5 (**CCR5**) gene has been described in subjects who remain uninfected despite extensive exposure to **HIV**-1. This allele was found to be common in the Caucasian population with a frequency of 0.0808, but was not found in people of African or Asian ancestry. To determine its role in **HIV**-1 transmission and disease progression, we analyzed the CCRS genotype of 1252 homosexual men enrolled in the Chicago component of the Multicenter AIDS Cohort Study (MACS). No infected participant was found to be homozygous for the delta 32 allele, whereas 3.6% of at-risk but uninfected Caucasian participants were homozygous, showing the highly protective role of this genotype against sexual acquisition of **HIV**-1. No evidence was found to suggest that heterozygotes were protected against **HIV**-1 infection, but a limited protective role against disease progression was noted. The delta 32 allele of **CCR5** is therefore an important host factor in **HIV**-1 transmission and pathogenesis.

L18 ANSWER 17 OF 36 MEDLINE on STN
97053783. PubMed ID: 8898197. Regions in beta-chemokine receptors **CCR5** and CCR2b that determine **HIV**-1 cofactor specificity. Rucker J; Samson M; Doranz B J; Libert F; Berson J F; Yi Y; Smyth R J; Collman R G; Broder C C; Vassart G; Doms R W; Parmentier M. (Department of Pathology and Laboratory Medicine, University of Pennsylvania, Philadelphia 19104, USA.) Cell, **(1996 Nov 1)** 87 (3) 437-46. Journal code: 0413066. ISSN: 0092-8674. Pub. country: United States. Language: English.
AB Macrophage-tropic (M-tropic) **HIV**-1 strains use the beta-chemokine receptor **CCR5**, but not CCR2b, as a cofactor for membrane fusion and infection, while the dual-tropic strain 89.6 uses both. **CCR5**/2b chimeras and mutants were used to map regions of **CCR5** important for cofactor function and specificity. M-tropic strains required either the amino-terminal domain or the first extracellular loop of **CCR5**. A CCR2b chimera containing the first 20 N-terminal residues of **CCR5** supported M-tropic envelope protein fusion. Amino-terminal truncations of **CCR5**/CCR2b chimeras indicated that residues 2-5 are important for M-tropic viruses, while 89.6 is dependent on residues 6-9. The identification of multiple functionally important regions in **CCR5**, coupled with differences in how **CCR5** is used by M- and dual-tropic viruses, suggests that interactions between **HIV**-1 and entry cofactors are conformationally complex.

L18 ANSWER 18 OF 36 MEDLINE on STN
97048157. PubMed ID: 8892998. **HIV**-1 subtype and second-receptor use. Zhang L; Huang Y; He T; Cao Y; Ho D D. Nature, **(1996 Oct 31)** 383 (6603) 768. Journal code: 0410462. ISSN: 0028-0836. Pub. country: ENGLAND: United Kingdom. Language: English.

L18 ANSWER 19 OF 36 MEDLINE on STN
96409665. PubMed ID: 8830405. **HIV** fusion. Bristow C L. Science, **(1996 Sep 20)** 273 (5282) 1642-3. Journal code: 0404511. ISSN: 0036-8075. Pub. country: United States. Language: English.

L18 ANSWER 20 OF 36 MEDLINE on STN
96406192. PubMed ID: 8815542. AIDS research. Receptor mutations help slow disease progression. Cohen J. Science, **(1996 Sep 27)** 273 (5283) 1797-8. Journal code: 0404511. ISSN: 0036-8075. Pub. country: United States. Language: English.

L18 ANSWER 21 OF 36 MEDLINE on STN

Wilkinson D. (Chester Beatty Laboratories, Institute of Cancer Research, 237 Fulham Road, London SW3 6JB UK.) Current biology : CB, **(1996 Sep 1)** 6 (9) 1051-3. Ref: 24. Journal code: 9107782. ISSN: 0960-9822. Pub. country: ENGLAND: United Kingdom. Language: English.

AB The identification of the cofactors required for **HIV**-1 entry into cells promises to provide new insights into viral transmission and pathogenesis, and opens new avenues for AIDS therapy and prophylaxis.

L18 ANSWER 22 OF 36 MEDLINE on STN

96386394. PubMed ID: 8791590. Genetic restriction of **HIV**-1 infection and progression to AIDS by a deletion allele of the **CKR5** structural gene. Hemophilia Growth and Development Study, Multicenter AIDS Cohort Study, Multicenter Hemophilia Cohort Study, San Francisco City Cohort, ALIVE Study. Dean M; Carrington M; Winkler C; Huttley G A; Smith M W; Allikmets R; Goedert J J; Buchbinder S P; Vittinghoff E; Gomperts E; Donfield S; Vlahov D; Kaslow R; Saah A; Rinaldo C; Detels R; O'Brien S J. (Laboratory of Genomic Diversity, National Cancer Institute (NCI), Frederick, MD 21702-1201, USA.) Science, **(1996 Sep 27)** 273 (5283) 1856-62. Journal code: 0404511. ISSN: 0036-8075. Pub. country: United States. Language: English.

AB The chemokine receptor 5 (**CKR5**) protein serves as a secondary receptor on CD4(+) T lymphocytes for certain strains of **human immunodeficiency virus**-type 1 (**HIV**-1). The **CKR5** structural gene was mapped to human chromosome 3p21, and a 32-base pair deletion allele (CKR5Delta32) was identified that is present at a frequency of approximately0.10 in the Caucasian population of the United States. An examination of 1955 patients included among six well-characterized acquired immunodeficiency syndrome (AIDS) cohort studies revealed that 17 deletion homozygotes occurred exclusively among 612 exposed **HIV**-1 antibody-negative individuals (2.8 percent) and not at all in 1343 **HIV**-1-infected individuals. The frequency of **CKR5** deletion heterozygotes was significantly elevated in groups of individuals that had survived **HIV**-1 infection for more than 10 years, and, in some risk groups, twice as frequent as their occurrence in rapid progressors to AIDS. Survival analysis clearly shows that disease progression is slower in **CKR5** deletion heterozygotes than in individuals homozygous for the normal **CKR5** gene. The CKR5Delta32 deletion may act as a recessive restriction gene against **HIV**-1 infection and may exert a dominant phenotype of delaying progression to AIDS among infected individuals.

L18 ANSWER 23 OF 36 MEDLINE on STN

96376339. PubMed ID: 8782446. Resistance to **HIV**-1 infection: it's in the genes. Fauci A S. (National Institute of Allergy and Infectious Diseases, National Institutes of Health Bethesda, Maryland 20892, USA.) Nature medicine, **(1996 Sep)** 2 (9) 966-7. Journal code: 9502015. ISSN: 1078-8956. Pub. country: United States. Language: English.

L18 ANSWER 24 OF 36 MEDLINE on STN

96345670. PubMed ID: 8751444. Resistance to **HIV**-1 infection in caucasian individuals bearing mutant alleles of the CCR-5 chemokine receptor gene. Samson M; Libert F; Doranz B J; Rucker J; Liesnard C; Farber C M; Saragosti S; Lapoumeroulie C; Cognaux J; Forceille C; Muyldermans G; Verhofstede C; Burtonboy G; Georges M; Imai T; Rana S; Yi Y; Smyth R J; Collman R G; Doms R W; Vassart G; Parmentier M. (IRIBHN and Services de Genetique Medicale, Virologie and Immunodeficiences, Universite Libre de Bruxelles, Belgium.) Nature, **(1996 Aug 22)** 382 (6593) 722-5. Journal code: 0410462. ISSN: 0028-0836. Pub. country: ENGLAND: United Kingdom. Language: English.

AB **HIV**-1 and related viruses require co-receptors, in addition to CD4, to infect target cells. The chemokine receptor CCR-5 (ref.1) was recently demonstrated to be a co-receptor for macrophage-tropic (M-tropic) **HIV**-1 strains, and the orphan receptor LESTR (also called fusin) allows infection by strains adapted for growth in transformed T-cell lines (T-tropic strains). Here we show that a mutant allele of CCR-5 is present at a high frequency in caucasian populations (allele frequency, 0.092), but is absent in black populations from Western and Central Africa and Japanese populations. A 32-base-pair deletion within the coding region results in a frame shift, and generates a non-functional receptor that does not support membrane fusion or infection by macrophage- and dual-tropic **HIV**-1 strains. In a cohort of **HIV**-1 infected caucasian subjects, no individual homozygous for the mutation was found, and the frequency of heterozygotes was 35% lower than in the general population. White blood cells from an individual homozygous for the null allele were found to be highly resistant to infection by M-tropic **HIV**-1 viruses, confirming that CCR-5 is the major co-receptor for primary **HIV**-1 strains. The lower frequency of heterozygotes in seropositive patients may indicate partial resistance.

L18 ANSWER 25 OF 36 MEDLINE on STN

96345657. PubMed ID: 8751431. Natural resistance to **HIV**?. Hill C M; Littman D R. Nature, **(1996 Aug 22)** 382 (6593) 668-9. Journal code: 0410462. ISSN: 0028-0836. Pub. country: ENGLAND: United Kingdom. Language:

L18 ANSWER 26 OF 36 MEDLINE on STN
96339434. PubMed ID: 8756719. Homozygous defect in **HIV**-1 coreceptor accounts for resistance of some multiply-exposed individuals to **HIV**-1 infection. Liu R; Paxton W A; Choe S; Ceradini D; Martin S R; Horuk R; MacDonald M E; Stuhlmann H; Koup R A; Landau N R. (Aaron Diamond AIDS Research Center, Rockefeller University New York, New York 10016, USA.) Cell, **(1996 Aug 9)** 86 (3) 367-77. Journal code: 0413066. ISSN: 0092-8674. Pub. country: United States. Language: English.

AB Rare individuals have been multiply exposed to **HIV**-1 but remain uninfected. The CD4+ T-cells of two of these individuals, designated EU2 and EU3, are highly resistant in vitro to the entry of primary macrophagetropic virus but are readily infectable with transformed T-cell line adapted viruses. We report here on the genetic basis of this resistance. We found that EU2 and EU3 have a homozygous defect in CKR-5, the gene encoding the recently described coreceptor for primary **HIV**-1 isolates. These individuals appear to have inherited a defective CKR-5 allele that contains an internal 32 base pair deletion. The encoded protein is severely truncated and cannot be detected at the cell surface. Surprisingly, this defect has no obvious phenotype in the affected individuals. Thus, a CKR-5 allele present in the human population appears to protect homozygous individuals from sexual transmission of **HIV**-1. Heterozygous individuals are quite common (approximately 20%) in some populations. These findings indicate the importance of CKR-5 in **HIV**-1 transmission and suggest that targeting the **HIV**-1-CKR-5 interaction may provide a means of preventing or slowing disease progression.

L18 ANSWER 27 OF 36 MEDLINE on STN
96332177. PubMed ID: 8709791. **HIV**-1-resistant individuals may lack **HIV**-1 coreceptor. Bradbury J. Lancet, **(1996 Aug 17)** 348 (9025) 463. Journal code: 2985213R. ISSN: 0140-6736. Pub. country: ENGLAND: United Kingdom. Language: English.

L18 ANSWER 28 OF 36 MEDLINE on STN
96320410. PubMed ID: 8685713. AIDS conference. Chemokines share center stage with drug therapies. Cohen J. Science, **(1996 Jul 19)** 273 (5273) 302-3. Journal code: 0404511. ISSN: 0036-8075. Pub. country: United States. Language: English.

L18 ANSWER 29 OF 36 MEDLINE on STN
96291862. PubMed ID: 8663314. Molecular cloning and functional characterization of a novel human CC chemokine receptor (**CCR5**) for RANTES, MIP-1beta, and MIP-1alpha. Raport C J; Gosling J; Schweickart V L; Gray P W; Charo I F. (ICOS Corporation, Bothell, Washington 98021, USA.) Journal of biological chemistry, **(1996 Jul 19)** 271 (29) 17161-6. Journal code: 2985121R. ISSN: 0021-9258. Pub. country: United States. Language: English.

AB Chemokines affect leukocyte chemotactic and activation activities through specific G protein-coupled receptors. In an effort to map the closely linked CC chemokine receptor genes, we identified a novel chemokine receptor encoded 18 kilobase pairs downstream of the monocyte chemoattractant protein-1 (MCP-1) receptor (CCR2) gene on human chromosome 3p21. The deduced amino acid sequence of this novel receptor, designated **CCR5**, is most similar to CCR2B, sharing 71% identical residues. Transfected cells expressing the receptor bind RANTES (regulated on activation normal T cell expressed), MIP-1beta, and MIP-1alpha with high affinity and generate inositol phosphates in response to these chemokines. This same combination of chemokines has recently been shown to potently inhibit **human immunodeficiency virus** replication in human peripheral blood leukocytes (Cocchi, F., DeVico, A. L., Garzino-Demo, A., Arya, S. K., Gallo, R. C., and Lusso, P.(1995) Science 270, 1811-1815). **CCR5** is expressed in lymphoid organs such as thymus and spleen, as well as in peripheral blood leukocytes, including macrophages and T cells, and is the first example of a human chemokine receptor that signals in response to MIP-1beta.

L18 ANSWER 30 OF 36 MEDLINE on STN
96275693. PubMed ID: 8658171. CC **CKR5**: a RANTES, MIP-1alpha, MIP-1beta receptor as a fusion cofactor for macrophage-tropic **HIV**-1. Alkhatib G; Combadiere C; Broder C C; Feng Y; Kennedy P E; Murphy P M; Berger E A. (Laboratory of Viral Diseases, National Institute of Allergy and Infectious Diseases (NIAID), National Institutes of Health (NIH), Bethesda, Maryland 20892, USA.) Science, **(1996 Jun 28)** 272 (5270) 1955-8. Journal code: 0404511. ISSN: 0036-8075. Pub. country: United States. Language: English.

AB **Human immunodeficiency virus**-type 1 (**HIV**-1) entry requires fusion cofactors on the CD4+ target cell. Fusin, a heterotrimeric GTP-binding protein (G protein)-coupled receptor, serves as a cofactor for T cell line-tropic isolates. The chemokines RANTES, MIP-1alpha, and MIP-1beta, which suppress infection by macrophage-tropic isolates, selectively inhibited cell fusion mediated by the corresponding envelope glycoproteins (Envs). Recombinant CC **CKR5**, a G protein-coupled receptor for these

preferentially with macrophage-tropic Envs. CC **CKR5** messenger RNA was detected selectively in cell types susceptible to macrophage-tropic isolates. CC **CKR5** is thus a fusion cofactor for macrophage-tropic **HIV**-1 strains.

L18 ANSWER 31 OF 36 MEDLINE on STN
96270516. PubMed ID: 8674120. A dual-tropic primary **HIV**-1 isolate that uses fusin and the beta-chemokine receptors CKR-5, CKR-3, and CKR-2b as fusion cofactors. Doranz B J; Rucker J; Yi Y; Smyth R J; Samson M; Peiper S C; Parmentier M; Collman R G; Doms R W. (Department of Pathology and Laboratory Medicine, University of Pennsylvania, Philadelphia 19104, USA.) Cell, **(1996 Jun 28)** 85 (7) 1149-58. Journal code: 0413066. ISSN: 0092-8674. Pub. country: United States. Language: English.
AB Here, we show that the beta-chemokine receptor CKR-5 serves as a cofactor for M-tropic **HIV** viruses. Expression of CKR-5 with CD4 enables nonpermissive cells to form syncytia with cells expressing M-tropic, but not T-tropic, **HIV**-1 env proteins. Expression of CKR-5 and CD4 enables entry of a M-tropic, but not a T-tropic, virus strain. A dual-tropic primary **HIV**-1 isolate (89.6) utilizes both Fusin and CKR-5 as entry cofactors. Cells expressing the 89.6 env protein form syncytia with QT6 cells expressing CD4 and either Fusin or CKR-5. The beta-chemokine receptors CKR-3 and CKR-2b support **HIV**-1 89.6 env-mediated syncytia formation but do not support fusion by any of the T-tropic or M-tropic strains tested. Our results suggest that the T-tropic viruses characteristic of disease progression may evolve from purely M-tropic viruses prevalent early in virus infection through changes in the env protein that enable the virus to use multiple entry cofactors.

L18 ANSWER 32 OF 36 MEDLINE on STN
96270515. PubMed ID: 8674119. The beta-chemokine receptors CCR3 and **CCR5** facilitate infection by primary **HIV**-1 isolates. Choe H; Farzan M; Sun Y; Sullivan N; Rollins B; Ponath P D; Wu L; Mackay C R; LaRosa G; Newman W; Gerard N; Gerard C; Sodroski J. (Division of Human Retrovirology Dana-Farber Cancer Institute, Boston, Massachusetts 02115, USA.) Cell, **(1996 Jun 28)** 85 (7) 1135-48. Journal code: 0413066. ISSN: 0092-8674. Pub. country: United States. Language: English.
AB We examined the ability of chemokine receptors and related G protein-coupled receptors to facilitate infection by primary, clinical **HIV**-1 isolates. **CCR5**, when expressed along with CD4, the **HIV**-1 receptor, allowed cell lines resistant to most primary **HIV**-1 isolates to be infected. CCR3 facilitated infection by a more restricted subset of primary viruses, and binding of the CCR3 ligand, eotaxin, inhibited infection by these isolates. Utilization of CCR3 and **CCR5** on the target cell depended upon the sequence of the third variable (V3) region of the **HIV**-1 gp120 exterior envelope glycoprotein. The ability of various members of the chemokine receptor family to support the early stages of **HIV**-1 infection helps to explain viral tropism and beta-chemokine inhibition of primary **HIV**-1 isolates.

L18 ANSWER 33 OF 36 MEDLINE on STN
96260018. PubMed ID: 8649512. **HIV**-1 entry into CD4+ cells is mediated by the chemokine receptor **CC-CKR-5**. Dragic T; Litwin V; Allaway G P; Martin S R; Huang Y; Nagashima K A; Cayanan C; Maddon P J; Koup R A; Moore J P; Paxton W A. (The Aaron Diamond AIDS Research Center, The Rockefeller University, New York 10016, USA.) Nature, **(1996 Jun 20)** 381 (6584) 667-73. Journal code: 0410462. ISSN: 0028-0836. Pub. country: ENGLAND: United Kingdom. Language: English.
AB The beta-chemokines MIP-1alpha, MIP-1beta and RANTES inhibit infection of CD4+ T cells by primary, non-syncytium-inducing (NSI) **HIV**-1 strains at the virus entry stage, and also block env-mediated cell-cell membrane fusion. CD4+ T cells from some **HIV**-1-exposed uninfected individuals cannot fuse with NSI **HIV**-1 strains and secrete high levels of beta-chemokines. Expression of the beta-chemokine receptor **CC-CKR-5** in CD4+, non-permissive human and non-human cells renders them susceptible to infection by NSI strains, and allows env-mediated membrane fusion. **CC-CKR-5** is a second receptor for NSI primary viruses.

L18 ANSWER 34 OF 36 MEDLINE on STN
96260017. PubMed ID: 8649511. Identification of a major co-receptor for primary isolates of **HIV**-1. Deng H; Liu R; Ellmeier W; Choe S; Unutmaz D; Burkhart M; Di Marzio P; Marmon S; Sutton R E; Hill C M; Davis C B; Peiper S C; Schall T J; Littman D R; Landau N R. (Skirball Institute for BioMolecular Medicine, New York University Medical Center, 10016, USA.) Nature, **(1996 Jun 20)** 381 (6584) 661-6. Journal code: 0410462. ISSN: 0028-0836. Pub. country: ENGLAND: United Kingdom. Language: English.
AB Entry of **HIV**-1 into target cells requires cell-surface CD4 and additional host cell cofactors. A cofactor required for infection with virus adapted for growth in transformed T-cell lines was recently identified and named fusin. However, fusin does not promote entry of macrophage-tropic viruses, which are believed to be the key pathogenic strains in vivo. The principal cofactor for entry mediated by the envelope glycoproteins of primary macrophage-tropic strains of **HIV**-1 is

MIP-1beta.

L18 ANSWER 35 OF 36 MEDLINE on STN
96260012. PubMed ID: 8649506. Hot fusion of **HIV**. Weiss R A; Clapham P R. Nature, **(1996 Jun 20)** 381 (6584) 647-8. Journal code: 0410462. ISSN: 0028-0836. Pub. country: ENGLAND: United Kingdom. Language: English.

L18 ANSWER 36 OF 36 MEDLINE on STN
96256468. PubMed ID: 8650568. A second coreceptor for **HIV** in early stages of infection. Balter M. Science, **(1996 Jun 21)** 272 (5269) 1740. Journal code: 0404511. ISSN: 0036-8075. Pub. country: United States. Language: English.

=> d his

(FILE 'HOME' ENTERED AT 13:34:37 ON 22 JAN 2006)

FILE 'USPATFULL' ENTERED AT 13:34:46 ON 22 JAN 2006
E ALLAWAY G P/IN
L1 22 S E4
E LITWIN V M/IN
L2 9 S E4
L3 1 S L2 NOT L1
E MADDON P J/IN
L4 47 S E4
L5 33 S L4 NOT (L1 OR L2)
L6 11 S L5 AND (CCR5 OR CC-CKR-5 OR CKR5)
L7 43461 S (HIV OR HUMAN IMMUNODEFICIENCY VIRUS)
L8 1714 S L7 AND (CCR5 OR CC-CKR-5 OR CKR5)
L9 10 S L8 AND AY<1997
L10 10 S L9 NOT L1

FILE 'MEDLINE' ENTERED AT 13:41:48 ON 22 JAN 2006
E ALLAWAY G P/AU
L11 28 S E3-E5
L12 4 S L11 AND (CCR5 OR CC-CKR-5 OR CKR5)
E LITWIN V M/AU
E MADDON P J/AU
L13 43 S E2-E4
L14 33 S L13 NOT L11
L15 3 S L14 AND (CCR5 OR CC-CKR-5 OR CKR5)
L16 160375 S (HIV OR HUMAN IMMUNODEFICIENCY VIRUS)
L17 2456 S L16 AND (CCR5 OR CC-CKR-5 OR CKR5)
L18 36 S L17 AND PY<1997

=> file wpids

COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	18.14	99.51

FILE 'WPIDS' ENTERED AT 13:45:59 ON 22 JAN 2006

FILE LAST UPDATED: 20 JAN 2006 <20060120/UP>
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http://www.stn-international.de/stndatabases/details/ipc_reform.html and

```
=> e allaway g p/in
E1            1     ALLAWAY E L/IN
E2            1     ALLAWAY G/IN
E3           22 --> ALLAWAY G P/IN
E4            1     ALLAWAY J B/IN
E5            1     ALLAWAY J R/IN
E6            5     ALLAWAY M B/IN
E7            1     ALLAWAY M J/IN
E8            1     ALLAWAY P/IN
E9            1     ALLAWAY P N/IN
E10           1     ALLAWAY S/IN
E11           1     ALLAWAY S M/IN
E12           3     ALLAWI H/IN

=> s e3
L19          22 "ALLAWAY G P"/IN

=> s l19 and (CCR5 or CC-CKR-5 or CKR5)
           426 CCR5
         23303 CC
            46 CKR
       4071028 5
             4 CC-CKR-5
                 (CC(W)CKR(W)5)
            10 CKR5
L20          6 L19 AND (CCR5 OR CC-CKR-5 OR CKR5)

=> d l20,bib,1-6

L20  ANSWER 1 OF 6  WPIDS  COPYRIGHT 2006 THE THOMSON CORP on STN
Full Text
AN   2004-389521 [36]   WPIDS
DNC  C2004-145921
TI   Identifying compounds useful for decreasing the ability of a virus, e.g.
     HIV-1 to infect previously uninfected cells comprises measuring the
     ability of the candidate compound to induce conformational changes in
     viral envelope glycoprotein.
DC   B04 D16
IN   ALLAWAY, G P; SALZWEDEL, K; WILD, C T
PA   (PANA-N) PANACOS PHARM INC
CYC  106
PI   WO 2004035808   A2 20040429 (200436)* EN   83
        RW: AT BE BG CH CY CZ DE DK EA EE ES FI FR GB GH GM GR HU IE IT KE LS
            LU MC MW MZ NL OA PT RO SD SE SI SK SL SZ TR TZ UG ZM ZW
         W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CO CR CU CZ DE DK
            DM DZ EC EE EG ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP
            KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NI NO NZ OM PG
            PH PL PT RO RU SC SD SE SG SK SL SY TJ TM TN TR TT TZ UA UG US UZ
            VC VN YU ZA ZM ZW
     US 2004132011   A1 20040708 (200445)
     AU 2003277378   A1 20040504 (200467)
ADT  WO 2004035808 A2 WO 2003-US32582 20031016; US 2004132011 A1 Provisional US
     2002-418341P 20021016, US 2003-685801 20031016; AU 2003277378 A1 AU
     2003-277378 20031016
FDT  AU 2003277378 A1 Based on WO 2004035808
PRAI US 2002-418341P      20021016; US 2003-685801       20031016

L20  ANSWER 2 OF 6  WPIDS  COPYRIGHT 2006 THE THOMSON CORP on STN
Full Text
AN   2002-215082 [27]   WPIDS
CR   1999-080861 [07]; 2000-571320 [53]; 2003-238109 [23]
DNC  C2002-065727
TI   Inhibiting human immunodeficiency virus 1 infection of a CD4+ cell by
     contacting the cell with antibody capable of binding to a chemokine
     receptor on the surface of the cell.
DC   B04 D16
IN   ALLAWAY, G P; LITWIN, V M; MADDON, P J; OLSON, W C
PA   (PROG-N) PROGENICS PHARM INC
CYC  1
PI   US 6344545      B1 20020205 (200227)*      25
ADT  US 6344545 B1 Provisional US 1996-14532P 19960402, Provisional US
     1996-19715P 19960614, US 1997-831823 19970402
PRAI US 1997-831823       19970402; US 1996-14532P       19960402;
     US 1996-19715P       19960614

L20  ANSWER 3 OF 6  WPIDS  COPYRIGHT 2006 THE THOMSON CORP on STN
Full Text
AN   2001-626098 [72]   WPIDS
DNC  C2001-186482
TI   Immunogenic composition for inhibiting HIV infection, comprises viral
     envelope protein or its fragment exterior to viral membrane, a stabilizing
```

DC B04 D16
IN **ALLAWAY, G P**; WILD, C T
PA (PANA-N) PANACOS PHARM INC; (ALLA-I) ALLAWAY G P; (WILD-I) WILD C T
CYC 96
PI WO 2001070262 A2 20010927 (200172)* EN 84
RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW MZ NL OA PT SD SE SL SZ TR TZ UG ZW
W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CO CR CU CZ DE DK DM DZ EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT TZ UA UG US UZ VN YU ZA ZW
AU 2001043639 A 20011003 (200210)
US 2002010317 A1 20020124 (200210)
EP 1267919 A2 20030102 (200310) EN
R: AL AT BE CH CY DE DK ES FI FR GB GR IE IT LI LT LU LV MC MK NL PT RO SE SI TR
ZA 2002008266 A 20031231 (200408) 100
NZ 521977 A 20040528 (200437)
ADT WO 2001070262 A2 WO 2001-US8108 20010315; AU 2001043639 A AU 2001-43639 20010315; US 2002010317 A1 Provisional US 2000-189981P 20000317, US 2001-809060 20010316; EP 1267919 A2 EP 2001-916641 20010315, WO 2001-US8108 20010315; ZA 2002008266 A ZA 2002-8266 20021014; NZ 521977 A NZ 2001-521977 20010315, WO 2001-US8108 20010315
FDT AU 2001043639 A Based on WO 2001070262; EP 1267919 A2 Based on WO 2001070262; NZ 521977 A Based on WO 2001070262
PRAI US 2000-189981P 20000317; US 2001-809060 20010316

L20 ANSWER 4 OF 6 WPIDS COPYRIGHT 2006 THE THOMSON CORP on STN
Full Text
AN 2000-571320 [53] WPIDS
CR 1999-080861 [07]; 2002-215082 [27]; 2003-238109 [23]
DNC C2000-170238
TI Determining an agent capable of inhibiting HIV-1 infection of a susceptible CD4+ cell comprises contacting a chemokine receptor and gp120/CD4+ complex in presence and absence of the agent and comparing them.
DC B04 D16
IN **ALLAWAY, G P**; LITWIN, V M; MADDON, P J; OLSON, W C
PA (PROG-N) PROGENICS PHARM INC
CYC 1
PI US 6107019 A 20000822 (200053)* 26
ADT US 6107019 A Provisional US 1996-14532P 19960402, Provisional US 1996-19715P 19960614, CIP of US 1997-831823 19970402, US 1997-876078 19970613
PRAI US 1997-876078 19970613; US 1996-14532P 19960402; US 1996-19715P 19960614; US 1997-831823 19970402

L20 ANSWER 5 OF 6 WPIDS COPYRIGHT 2006 THE THOMSON CORP on STN
Full Text
AN 1998-086551 [08] WPIDS
CR 1998-086550 [08]
DNC C1998-029219
TI Chemokine receptor **CCR5** fragments - useful for inhibition of Human Immunodeficiency Virus 1 infection.
DC B04 D16
IN **ALLAWAY, G P**; DRAGIC, T; LITWIN, V M; MADDON, P J; MOORE, J P; TRKOLA, A
PA (AARO-N) AARON DIAMOND AIDS RES CENT; (PROG-N) PROGENICS PHARM INC; (ADAR-N) ADARC AARON DIAMOND AIDS RES CENT
CYC 22
PI WO 9747319 A1 19971218 (199808)* EN 106
RW: AT BE CH DE DK ES FI FR GB GR IE IT LU MC NL PT SE
W: AU CA JP MX
AU 9734026 A 19980107 (199820)
EP 956044 A1 19991117 (199953) EN
R: AT BE CH DE DK ES FI FR GB GR IE IT LI LU MC NL PT SE
JP 2001503608 W 20010321 (200122) 92
MX 9810425 A1 20000501 (200129)
AU 735460 B 20010712 (200147)
ADT WO 9747319 A1 WO 1997-US10619 19970613; AU 9734026 A AU 1997-34026 19970613; EP 956044 A1 EP 1997-930120 19970613, WO 1997-US10619 19970613; JP 2001503608 W WO 1997-US10619 19970613, JP 1998-501895 19970613; MX 9810425 A1 MX 1998-10425 19981209; AU 735460 B AU 1997-34026 19970613
FDT AU 9734026 A Based on WO 9747319; EP 956044 A1 Based on WO 9747319; JP 2001503608 W Based on WO 9747319; AU 735460 B Previous Publ. AU 9734026, Based on WO 9747319
PRAI US 1996-665090 19960614; US 1996-19941P 19960614

L20 ANSWER 6 OF 6 WPIDS COPYRIGHT 2006 THE THOMSON CORP on STN
Full Text
AN 1998-086550 [08] WPIDS
CR 1998-086551 [08]
DNC C1998-029218

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     Immunodeficiency Virus 1 infection.
DC   B04 D16
IN   ALLAWAY, G P; DRAGIC, T; LITWIN, V M; MADDON, P J; MOORE, J P; TRKOLA, A
PA   (AARO-N) AARON DIAMOND AIDS RES CENT; (PROG-N) PROGENICS PHARM INC;
     (ADAR-N) ADARC AARON DIAMOND AIDS RES CENT
CYC  22
PI   WO 9747318      A1 19971218 (199808)* EN   86
        RW: AT BE CH DE DK ES FI FR GB GR IE IT LU MC NL PT SE
         W: AU CA JP MX
     AU 9733902      A  19980107 (199820)
     AU 2001076114   A  20011206 (200208)#
     US 2004086528   A1 20040506 (200430)
     AU 2004205143   A1 20040916 (200479)#
ADT  WO 9747318 A1 WO 1997-US10233 19970613; AU 9733902 A AU 1997-33902
     19970613; AU 2001076114 A Div ex AU 1997-34026 19970613, AU 2001-76114
     20010926; US 2004086528 A1 Provisional US 1996-19941P 19960614, Cont of US
     1997-874618 19970613, Cont of US 2000-724105 20001128, US 2001-852238
     20010509; AU 2004205143 A1 Div ex AU 2001-76114 20010926, AU 2004-205143
     20040819
FDT  AU 9733902 A Based on WO 9747318; AU 2001076114 A Div ex AU 735460
PRAI US 1996-665090       19960614; US 1996-19941P       19960614;
     AU 2001-76114        20010926; US 1997-874618       19970613;
     US 2000-724105       20001128; US 2001-852238       20010509;
     AU 2004-205143       20040819


=> d his

     (FILE 'HOME' ENTERED AT 13:34:37 ON 22 JAN 2006)

     FILE 'USPATFULL' ENTERED AT 13:34:46 ON 22 JAN 2006
                E ALLAWAY G P/IN
L1           22 S E4
                E LITWIN V M/IN
L2            9 S E4
L3            1 S L2 NOT L1
                E MADDON P J/IN
L4           47 S E4
L5           33 S L4 NOT (L1 OR L2)
L6           11 S L5 AND (CCR5 OR CC-CKR-5 OR CKR5)
L7        43461 S (HIV OR HUMAN IMMUNODEFICIENCY VIRUS)
L8         1714 S L7 AND (CCR5 OR CC-CKR-5 OR CKR5)
L9           10 S L8 AND AY<1997
L10          10 S L9 NOT L1

     FILE 'MEDLINE' ENTERED AT 13:41:48 ON 22 JAN 2006
                E ALLAWAY G P/AU
L11          28 S E3-E5
L12           4 S L11 AND (CCR5 OR CC-CKR-5 OR CKR5)
                E LITWIN V M/AU
                E MADDON P J/AU
L13          43 S E2-E4
L14          33 S L13 NOT L11
L15           3 S L14 AND (CCR5 OR CC-CKR-5 OR CKR5)
L16      160375 S (HIV OR HUMAN IMMUNODEFICIENCY VIRUS)
L17        2456 S L16 AND (CCR5 OR CC-CKR-5 OR CKR5)
L18          36 S L17 AND PY<1997

     FILE 'WPIDS' ENTERED AT 13:45:59 ON 22 JAN 2006
                E ALLAWAY G P/IN
L19          22 S E3
L20           6 S L19 AND (CCR5 OR CC-CKR-5 OR CKR5)

=> e litwin v m/in
E1            1     LITWIN S M/IN
E2            1     LITWIN STASZEWSKA E/IN
E3           10 --> LITWIN V M/IN
E4            3     LITWIN W/IN
E5            1     LITWIN W S/IN
E6            1     LITWIN Y/IN
E7            1     LITWINCHUK A/IN
E8            1     LITWINENKO A S/IN
E9            1     LITWINETZ D M/IN
E10           4     LITWING E/IN
E11           1     LITWINIUK J/IN
E12           1     LITWINOWI M J/IN

=> s e3
L21          10 "LITWIN V M"/IN

=> s l20 not l19
L22           0 L20 NOT L19
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=> e maddon p j/in
E1            9     MADDOCKS S/IN
E2            5     MADDOCKS T C/IN
E3           46 --> MADDON P J/IN
E4            1     MADDONA P/IN
E5            1     MADDONADO P/IN
E6            1     MADDOUX D/IN
E7            3     MADDOX A D/IN
E8            1     MADDOX A F/IN
E9            2     MADDOX B L/IN
E10           3     MADDOX C/IN
E11           7     MADDOX C A/IN
E12           2     MADDOX C B/IN

=> s e3-e4
            46 "MADDON P J"/IN
             1 "MADDONA P"/IN
L23         47 ("MADDON P J"/IN OR "MADDONA P"/IN)

=> s l23 not l19
L24         33 L23 NOT L19

=> s l24 and (CCR5 or CC-CKR-5 or CKR5)
           426 CCR5
         23303 CC
            46 CKR
       4071028 5
             4 CC-CKR-5
                 (CC(W)CKR(W)5)
            10 CKR5
L25          7 L24 AND (CCR5 OR CC-CKR-5 OR CKR5)

=> d l25,bib,1-7

L25  ANSWER 1 OF 7  WPIDS  COPYRIGHT 2006 THE THOMSON CORP on STN
Full Text
AN   2004-190050 [18]   WPIDS
DNC  C2004-074893
TI   Novel anti-CCR5 antibody useful for treating HIV, comprising two light
     chains, and two heavy chains, which binds to CCR5 on surface of human
     cell..
DC   A96 B04 D16
IN   HINTON, P R; MADDON, P J; OLSON, W C; TSURUSHITA, N; VASQUEZ, M
PA   (HINT-I) HINTON P R; (MADD-I) MADDON P J; (OLSO-I) OLSON W C; (TSUR-I)
     TSURUSHITA N; (VASQ-I) VASQUEZ M
CYC  1
PI   US 2003228306   A1 20031211 (200418)*      52
ADT  US 2003228306 A1 CIP of US 2002-358886 20020222, US 2003-371483 20030221
PRAI US 2003-371483       20030221; US 2002-358886       20020222

L25  ANSWER 2 OF 7  WPIDS  COPYRIGHT 2006 THE THOMSON CORP on STN
Full Text
AN   2003-803830 [75]   WPIDS
DNC  C2003-221841
TI   New anti-CCR5 antibody, useful for preparing a composition for treating
     or preventing Human Immunodeficiency Virus-1 infection.
DC   A96 B04 D16
IN   MADDON, P J; OLSON, W C; HINTON, P R; TSURUSHITA, N; VASQUEZ, M
PA   (PROG-N) PROGENICS PHARM INC; (PROT-N) PROTEIN DESIGN LABS INC
CYC  103
PI   WO 2003072766   A1 20030904 (200375)* EN  124
        RW: AT BE BG CH CY CZ DE DK EA EE ES FI FR GB GH GM GR HU IE IT KE LS
            LU MC MW MZ NL OA PT SD SE SI SK SL SZ TR TZ UG ZM ZW
         W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CO CR CU CZ DE DK
            DM DZ EC EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR
            KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NO NZ OM PH PL PT
            RO RU SC SD SE SG SK SL TJ TM TN TR TT TZ UA UG US UZ VC VN YU ZA
            ZM ZW
     AU 2003217674   A1 20030909 (200428)
     EP 1478738      A1 20041124 (200477)  EN
         R: AL AT BE BG CH CY CZ DE DK EE ES FI FR GB GR HU IE IT LI LT LU LV
            MC MK NL PT RO SE SI SK TR
     NO 2004003971   A  20041116 (200519)
     KR 2005004784   A  20050112 (200535)
ADT  WO 2003072766 A1 WO 2003-US5500 20030221; AU 2003217674 A1 AU 2003-217674
     20030221; EP 1478738 A1 EP 2003-713632 20030221, WO 2003-US5500 20030221;
     NO 2004003971 A WO 2003-US5500 20030221, NO 2004-3971 20040922; KR
     2005004784 A KR 2004-713080 20040820
FDT  AU 2003217674 A1 Based on WO 2003072766; EP 1478738 A1 Based on WO
     2003072766
PRAI US 2002-81128        20020222
```

Full Text
AN 2003-439416 [41] WPIDS
DNC C2003-116366
TI Reducing a subject's HIV-1 viral load, useful for treating HIV infection, comprises administering a viral load reducing amount of an antibody which binds to a **CCR5** chemokine receptor and which inhibits fusion of HIV-1 to a CD4+**CCR5**+ cell.
DC B04 D16
IN **MADDON, P J**; OLSON, W C
PA (MADD-I) MADDON P J; (OLSO-I) OLSON W C
CYC 1
PI US 2003044411 A1 20030306 (200341)* 50
ADT US 2003044411 A1 Provisional US 2001-282380P 20010406, US 2002-116797 20020405
PRAI US 2001-282380P 20010406; US 2002-116797 20020405

L25 ANSWER 4 OF 7 WPIDS COPYRIGHT 2006 THE THOMSON CORP on STN
Full Text
AN 2003-237965 [23] WPIDS
DNC C2003-060885
TI Reducing HIV infected subject's HIV-1 viral load, by administering antibody which binds to **CCR5** chemokine receptor and inhibits fusion of HIV-1 to CD4+**CCR5**+ cell.
DC B04 D16 P83
IN **MADDON, P J**; OLSON, W C
PA (MADD-I) MADDON P J; (OLSO-I) OLSON W C; (PROG-N) PROGENICS PHARM INC
CYC 100
PI US 2002146415 A1 20021010 (200323)* 47
WO 2002083172 A1 20021024 (200323) EN
RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW MZ NL OA PT SD SE SL SZ TR TZ UG ZM ZW
W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CO CR CU CZ DE DK DM DZ EC EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NO NZ OM PH PL PT RO RU SD SE SG SI SK SL TJ TM TN TR TT TZ UA UG UZ VN YU ZA ZM ZW
AU 2002303251 A1 20021028 (200433)
ADT US 2002146415 A1 US 2001-828615 20010406; WO 2002083172 A1 WO 2002-US10752 20020405; AU 2002303251 A1 AU 2002-303251 20020405
FDT AU 2002303251 A1 Based on WO 2002083172
PRAI US 2001-828615 20010406

L25 ANSWER 5 OF 7 WPIDS COPYRIGHT 2006 THE THOMSON CORP on STN
Full Text
AN 2002-362300 [39] WPIDS
CR 2001-483270 [52]
DNC C2002-102537
TI Composition for inhibiting human immunodeficiency virus-1 infection comprises mixture of three compounds which bind to **CCR5** receptor, retard attachment of HIV-1 to CD4+ cell, or retard gp41 from mediating fusion of HIV-1 to CD4+ cell.
DC B04 D16
IN **MADDON, P J**; OLSON, W C
PA (PROG-N) PROGENICS PHARM INC; (MADD-I) MADDON P J; (OLSO-I) OLSON W C
CYC 98
PI WO 2002022077 A2 20020321 (200239)* EN 147
RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW MZ NL OA PT SD SE SL SZ TR TZ UG ZW
W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CO CR CU CZ DE DK DM DZ EC EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NO NZ PH PL PT RO RU SD SE SG SI SK SL TJ TM TR TT TZ UA UG UZ VN YU ZA ZW
AU 2001090925 A 20020326 (200251)
US 2002106374 A1 20020808 (200254)
EP 1322332 A2 20030702 (200344) EN
R: AL AT BE CH CY DE DK ES FI FR GB GR IE IT LI LT LU LV MC MK NL PT RO SE SI TR
JP 2004518624 W 20040624 (200442) 220
ADT WO 2002022077 A2 WO 2001-US28756 20010914; AU 2001090925 A AU 2001-90925 20010914; US 2002106374 A1 CIP of US 2000-663219 20000915, CIP of WO 2001-US2633 20010126, Provisional US 2001-266738P 20010206, US 2001-912824 20010725; EP 1322332 A2 EP 2001-970984 20010914, WO 2001-US28756 20010914; JP 2004518624 W WO 2001-US28756 20010914, JP 2002-526332 20010914
FDT AU 2001090925 A Based on WO 2002022077; EP 1322332 A2 Based on WO 2002022077; JP 2004518624 W Based on WO 2002022077
PRAI US 2001-912824 20010725; US 2000-663219 20000915; WO 2001-US2633 20010126; US 2001-266738P 20010206

L25 ANSWER 6 OF 7 WPIDS COPYRIGHT 2006 THE THOMSON CORP on STN
Full Text
AN 2001-122993 [13] WPIDS
DNN N2001-090325 DNC C2001-035684
TI New viral envelope proteins, useful for producing vaccines to treat human

mutations such that viral transmembrane-surface protein complex is more stable.

DC B04 D16 S03

IN BINLEY, J M; **MADDON, P J**; MOORE, J P; OLSON, W C; SCHUELKE, N

PA (AARO-N) AARON DIAMOND AIDS RES CENT; (PROG-N) PROGENICS PHARM INC

CYC 23

PI WO 2001000648 A1 20010104 (200113)* EN 109
RW: AT BE CH CY DE DK ES FI FR GB GR IE IT LU MC NL PT SE
W: AU CA JP MX
AU 2000058842 A 20010131 (200124)
EP 1198468 A1 20020424 (200235) EN
R: AT BE CH CY DE DK ES FI FR GB GR IE IT LI LU MC NL PT SE
JP 2003509013 W 20030311 (200319) 97
AU 782123 B2 20050707 (200551)

ADT WO 2001000648 A1 WO 2000-US17267 20000623; AU 2000058842 A AU 2000-58842 20000623; EP 1198468 A1 EP 2000-944801 20000623, WO 2000-US17267 20000623; JP 2003509013 W WO 2000-US17267 20000623, JP 2001-507055 20000623; AU 782123 B2 AU 2000-58842 20000623

FDT AU 2000058842 A Based on WO 2001000648; EP 1198468 A1 Based on WO 2001000648; JP 2003509013 W Based on WO 2001000648; AU 782123 B2 Previous Publ. AU 2000058842, Based on WO 2001000648

PRAI US 1999-340992 19990625

L25 ANSWER 7 OF 7 WPIDS COPYRIGHT 2006 THE THOMSON CORP on STN

Full Text

AN 2000-431480 [37] WPIDS

DNC C2000-131148

TI Preventing and treating human immunodeficiency virus (HIV) infections using compounds that inhibit interactions between HIV and its fusion co-receptor, especially antibodies specific for the **CCR5** chemokine receptor.

DC B04 D16

IN **MADDON, P J**; OLSON, W C

PA (PROG-N) PROGENICS PHARM INC

CYC 30

PI WO 2000035409 A2 20000622 (200037)* EN 68
RW: AT BE CH CY DE DK ES FI FR GB GR IE IT LU MC NL PT SE
W: AU CA JP MX
AU 2000021996 A 20000703 (200046)
EP 1144006 A2 20011017 (200169) EN
R: AL AT BE CH CY DE DK ES FI FR GB GR IE IT LI LT LU LV MC MK NL PT RO SE SI
JP 2002538771 W 20021119 (200281) 65
MX 2001006097 A1 20020501 (200368)
AU 773175 B2 20040520 (200462)
US 2004228869 A1 20041118 (200477)
AU 2004205164 A1 20040916 (200479)
AU 2004205165 A1 20040916 (200479)

ADT WO 2000035409 A2 WO 1999-US30345 19991216; AU 2000021996 A AU 2000-21996 19991216; EP 1144006 A2 EP 1999-966466 19991216, WO 1999-US30345 19991216; JP 2002538771 W WO 1999-US30345 19991216, JP 2000-587730 19991216; MX 2001006097 A1 WO 1999-US30345 19991216, MX 2001-6097 20010615; AU 773175 B2 AU 2000-21996 19991216; US 2004228869 A1 Provisional US 1998-112532P 19981216, CIP of US 1999-464902 19991216, Cont of US 2000-594983 20000615, US 2004-763545 20040123; AU 2004205164 A1 AU 2004-205164 20040820; AU 2004205165 A1 AU 2004-205165 20040820

FDT AU 2000021996 A Based on WO 2000035409; EP 1144006 A2 Based on WO 2000035409; JP 2002538771 W Based on WO 2000035409; MX 2001006097 A1 Based on WO 2000035409; AU 773175 B2 Previous Publ. AU 2000021996, Based on WO 2000035409; AU 2004205164 A1 Div ex AU 773175; AU 2004205165 A1 Div ex AU 773175

PRAI US 1998-212793 19981216; US 1998-112532P 19981216;
US 1999-464902 19991216; US 2000-594983 20000615;
US 2004-763545 20040123

=> d his

(FILE 'HOME' ENTERED AT 13:34:37 ON 22 JAN 2006)

FILE 'USPATFULL' ENTERED AT 13:34:46 ON 22 JAN 2006

E ALLAWAY G P/IN

L1 22 S E4

E LITWIN V M/IN

L2 9 S E4

L3 1 S L2 NOT L1

E MADDON P J/IN

L4 47 S E4

L5 33 S L4 NOT (L1 OR L2)

L6 11 S L5 AND (CCR5 OR CC-CKR-5 OR CKR5)

L7 43461 S (HIV OR HUMAN IMMUNODEFICIENCY VIRUS)

L8 1714 S L7 AND (CCR5 OR CC-CKR-5 OR CKR5)

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L10          10 S L9 NOT L1

     FILE 'MEDLINE' ENTERED AT 13:41:48 ON 22 JAN 2006
                E ALLAWAY G P/AU
L11          28 S E3-E5
L12           4 S L11 AND (CCR5 OR CC-CKR-5 OR CKR5)
                E LITWIN V M/AU
                E MADDON P J/AU
L13          43 S E2-E4
L14          33 S L13 NOT L11
L15           3 S L14 AND (CCR5 OR CC-CKR-5 OR CKR5)
L16      160375 S (HIV OR HUMAN IMMUNODEFICIENCY VIRUS)
L17        2456 S L16 AND (CCR5 OR CC-CKR-5 OR CKR5)
L18          36 S L17 AND PY<1997

     FILE 'WPIDS' ENTERED AT 13:45:59 ON 22 JAN 2006
                E ALLAWAY G P/IN
L19          22 S E3
L20           6 S L19 AND (CCR5 OR CC-CKR-5 OR CKR5)
                E LITWIN V M/IN
L21          10 S E3
L22           0 S L20 NOT L19
                E MADDON P J/IN
L23          47 S E3-E4
L24          33 S L23 NOT L19
L25           7 S L24 AND (CCR5 OR CC-CKR-5 OR CKR5)

=> s (HIV or human immunodeficiency virus)
         21245 HIV
        173944 HUMAN
          7711 IMMUNODEFICIENCY
         41982 VIRUS
          4957 HUMAN IMMUNODEFICIENCY VIRUS
                 (HUMAN(W)IMMUNODEFICIENCY(W)VIRUS)
L26      21982 (HIV OR HUMAN IMMUNODEFICIENCY VIRUS)

=> s l26 and (CCR5 or CC-CKR-5 or CKR5)
           426 CCR5
         23303 CC
            46 CKR
       4071028 5
             4 CC-CKR-5
                 (CC(W)CKR(W)5)
            10 CKR5
L27         353 L26 AND (CCR5 OR CC-CKR-5 OR CKR5)

=> s l27 and ay<1997

SEARCH ENDED BY USER

=>
SEARCH ENDED BY USER

=> s l27 and ay=1995
         710099 AY=1995
                 (AY=1995)
L28           0 L27 AND AY=1995

=> s l27 and ay=1996
         753966 AY=1996
                 (AY=1996)
L29          12 L27 AND AY=1996

=> s l29 not l19
L30           9 L29 NOT L19

=> d l30,bib,1-9

L30  ANSWER 1 OF 9  WPIDS  COPYRIGHT 2006 THE THOMSON CORP on STN
Full Text
AN   2004-247705 [23]   WPIDS
CR   2003-707284 [67]
DNC  C2004-096675
TI   New isolated human immunodeficiency virus envelope polypeptides and
     oligonucleotides encoding the gp120 glycoproteins, useful for diagnosing,
     preventing or treating human immunodeficiency virus (HIV) infection.
DC   B04 D16
IN   BERMAN, P W
PA   (GETH) GENENTECH INC
CYC  1
PI   US 2004052821   A1 20040318 (200423)*     126
ADT  US 2004052821 A1 Provisional US 1996-69891P 19960708, Div ex US
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20030221
FDT US 2004052821 A1 Div ex US 6090392
PRAI US 1996-69891P 19960708; US 1997-889841 19970708;
US 1999-419362 19991015; US 2003-371472 20030221

L30 ANSWER 2 OF 9 WPIDS COPYRIGHT 2006 THE THOMSON CORP on STN
Full Text
AN 2004-118899 [12] WPIDS
CR 1998-018223 [02]; 2001-417127 [44]
DNN N2004-095034 DNC C2004-047617
TI Detecting the presence or activity of a translocation promoting agent, comprises contacting sample from a mammal with a binding partner of the promoting agent and detecting whether binding has occurred between the agent and partner.
DC B04 D16 S03
IN DENG, H; ELLMEIER, W; LANDAU, N R; LITTMAN, D R; LIU, R
PA (DENG-I) DENG H; (ELLM-I) ELLMEIER W; (LAND-I) LANDAU N R; (LITT-I) LITTMAN D R; (LIUR-I) LIU R
CYC 1
PI US 2003096221 A1 20030522 (200412)* 41
ADT US 2003096221 A1 **Provisional US 1996-17157P 19960520, Provisional US 1996-20043P 19960619,** CIP of US 1997-858660 19970519, Cont of US 1997-861105 19970521, US 2000-734221 20001211
FDT US 2003096221 A1 Cont of US 6258527
PRAI US 2000-734221 20001211; US 1996-17157P 19960520;
US 1996-20043P 19960619; US 1997-858660 19970519;
US 1997-861105 19970521

L30 ANSWER 3 OF 9 WPIDS COPYRIGHT 2006 THE THOMSON CORP on STN
Full Text
AN 2003-898109 [82] WPIDS
CR 1998-272141 [24]
DNC C2003-255145
TI New antibody that binds mammalian CC-chemokine receptor-5, useful e.g. for inhibiting infection by human immune deficiency virus or binding of chemokine.
DC B04 D16
IN MACKAY, C R; WU, L
PA (MILL-N) MILLENNIUM PHARM INC
CYC 1
PI US 2003166870 A1 20030904 (200382)* 43
ADT US 2003166870 A1 **CIP of US 1996-739507 19961028,** CIP of US 1997-893911 19970711, US 2001-870932 20010530
FDT US 2003166870 A1 CIP of US 6528625
PRAI US 2001-870932 20010530; US 1996-739507 19961028;
US 1997-893911 19970711

L30 ANSWER 4 OF 9 WPIDS COPYRIGHT 2006 THE THOMSON CORP on STN
Full Text
AN 2001-616350 [71] WPIDS
CR 1998-008876 [01]
DNC C2001-184510
TI Composition for preventing **HIV** infection of mammalian cells comprises an anti-immunodeficiency virus immunokine capable of binding to a cellular protein by which **HIV** infection of the cell is prevented.
DC B04 D16
IN MUNDSCHENK, D D; REID, P F
PA (PHYL-N) PHYLOMED CORP; (MUND-I) MUNDSCHENK D D; (REID-I) REID P F
CYC 96
PI WO 2001070173 A2 20010927 (200171)* EN 54
RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW MZ NL OA PT SD SE SL SZ TR TZ UG ZW
W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CO CR CU CZ DE DK DM DZ EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT TZ UA UG US UZ VN YU ZA ZW
AU 2001049194 A 20011003 (200210)
EP 1272512 A2 20030108 (200311) EN
R: AL AT BE CH CY DE DK ES FI FR GB GR IE IT LI LT LU LV MC MK NL PT RO SE SI TR
US 2003211465 A1 20031113 (200382)
ADT WO 2001070173 A2 WO 2001-US8150 20010314; AU 2001049194 A AU 2001-49194 20010314; EP 1272512 A2 EP 2001-922384 20010314, WO 2001-US8150 20010314; US 2003211465 A1 **Cont of US 1996-644399 19960510,** Cont of US 1997-908212 19970807, CIP of US 1999-368834 19990805, Div ex US 2000-533454 20000323, US 2002-292164 20021112
FDT AU 2001049194 A Based on WO 2001070173; EP 1272512 A2 Based on WO 2001070173; US 2003211465 A1 Cont of US 5989857
PRAI US 2000-533454 20000323; US 1996-644399 19960510;
US 1997-908212 19970807; US 1999-368834 19990805;
US 2002-292164 20021112

Full Text
AN 2001-417127 [44] WPIDS
CR 1998-018223 [02]; 2004-118899 [12]
DNN N2001-309041 DNC C2001-125944
TI Transformed mammalian cell (I) that contains a CD4 gene, reporter gene and **HIV** LTR for identification of drugs and antibodies for treatment of **HIV**.
DC B04 D16 S03
IN DENG, H; ELLMEIER, W; LANDAU, N R; LITTMAN, D R; LIU, R
PA (AARO-N) AARON DIAMOND AIDS RES CENT; (UYNY) UNIV NEW YORK STATE
CYC 1
PI US 6258527 B1 20010710 (200144)* 37
ADT US 6258527 B1 **Provisional US 1996-17157P 19960520, Provisional US 1996-20043P 19960619**, CIP of US 1997-858660 19970519, US 1997-861105 19970521
PRAI US 1997-861105 19970521; US 1996-17157P 19960520; US 1996-20043P 19960619; US 1997-858660 19970519

L30 ANSWER 6 OF 9 WPIDS COPYRIGHT 2006 THE THOMSON CORP on STN
Full Text
AN 1998-272141 [24] WPIDS
CR 2003-898109 [82]
DNN N1998-213636 DNC C1998-084982
TI Antibody that binds to mammalian cytokine receptor 5 - particularly for treating or preventing infection by human immune deficiency virus, also for diagnosing susceptibility to infection and to identify modulators of the receptor.
DC B04 D16 S03
IN MACKAY, C R; WU, L
PA (LEUK-N) LEUKOSITE INC; (MILL-N) MILLENNIUM PHARM INC
CYC 78
PI WO 9818826 A2 19980507 (199824)* EN 117
RW: AT BE CH DE DK EA ES FI FR GB GH GR IE IT KE LS LU MC MW NL OA PT SD SE SZ UG ZW
W: AL AM AT AU AZ BA BB BG BR BY CA CH CN CU CZ DE DK EE ES FI GB GE GH HU IL IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MD MG MK MN MW MX NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT UA UG UZ VN YU ZW
AU 9851553 A 19980522 (199840)
US 6528625 B1 20030304 (200320)
ADT WO 9818826 A2 WO 1997-US19661 19971027; AU 9851553 A AU 1998-51553 19971027; US 6528625 B1 **CIP of US 1996-739507 19961028**, US 1997-893911 19970711
FDT AU 9851553 A Based on WO 9818826
PRAI US 1997-893911 19970711; US 1996-739507 19961028

L30 ANSWER 7 OF 9 WPIDS COPYRIGHT 2006 THE THOMSON CORP on STN
Full Text
AN 1998-195463 [18] WPIDS
DNN N1998-154747 DNC C1998-062545
TI New isolated mouse chemokine receptor, CC-**CKR5** - used to develop products for the study, diagnosis and treatment of **HIV** infection or T-cell mediated inflammation.
DC B04 D16 P14 S03
IN BERGSMA, D J; BRAWNER, M E; SHABON, U
PA (SMIK) SMITHKLINE BEECHAM CORP
CYC 20
PI EP 834564 A2 19980408 (199818)* EN 27
R: AT BE CH DE DK ES FI FR GB GR IE IT LI LU MC NL PT SE
JP 10179180 A 19980707 (199837) 70
US 6388055 B1 20020514 (200239)
ADT EP 834564 A2 EP 1997-307823 19971003; JP 10179180 A JP 1997-307784 19971003; US 6388055 B1 **US 1996-724984 19961003**
PRAI US 1996-724984 19961003

L30 ANSWER 8 OF 9 WPIDS COPYRIGHT 2006 THE THOMSON CORP on STN
Full Text
AN 1998-032650 [03] WPIDS
DNN N1998-026158 DNC C1998-011136
TI CC chemokine receptor 5 polypeptide - used to inhibit membrane fusion between **HIV** and a target cell.
DC B04 D16 P14 S03
IN ALKHATIB, G; BERGER, E A; BRODER, C C; COMBADIERE, C; FENG, Y; KENNEDY, P E; MURPHY, P M
PA (USSH) US DEPT HEALTH & HUMAN SERVICES; (USSH) US DEP HEALTH & HUMAN SERVICES
CYC 77
PI WO 9745543 A2 19971204 (199803)* EN 70
RW: AT BE CH DE DK EA ES FI FR GB GH GR IE IT KE LS LU MC MW NL OA PT SD SE SZ UG
W: AL AM AT AU AZ BA BB BG BR BY CA CH CN CU CZ DE DK EE ES FI GB GE GH HU IL IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MD MG MK MN MW MX NO NZ PL PT RO RU SD SE SG SI SK TJ TM TR TT UA UG US UZ VN YU

EP 975749 A2 20000202 (200011) EN
R: AT BE CH DE DK ES FI FR GB GR IE IT LI LU MC NL PT SE
US 2003195348 A1 20031016 (200369)
US 2004259785 A1 20041223 (200504)
US 2005118677 A1 20050602 (200537)
ADT WO 9745543 A2 WO 1997-US9586 19970528; AU 9733756 A AU 1997-33756 19970528; EP 975749 A2 EP 1997-929777 19970528, WO 1997-US9586 19970528; US 2003195348 A1 **Provisional US 1996-18508P 19960528,** Cont of US 1997-864458 19970528, US 2003-439845 20030515; US 2004259785 A1 **Provisional US 1996-18508P 19960528,** Cont of US 1997-864458 19970528, US 2003-700313 20031031; US 2005118677 A1 **Provisional US 1996-18508P 19960528,** Cont of US 1997-864458 19970528, US 2004-846185 20040514
FDT AU 9733756 A Based on WO 9745543; EP 975749 A2 Based on WO 9745543
PRAI US 1996-18508P 19960528; US 1997-864458 19970528;
US 2003-439845 20030515; US 2003-700313 20031031;
US 2004-846185 20040514

L30 ANSWER 9 OF 9 WPIDS COPYRIGHT 2006 THE THOMSON CORP on STN
Full Text
AN 1998-018223 [02] WPIDS
CR 2001-417127 [44]; 2004-118899 [12]
DNN N1998-013884 DNC C1998-006768
TI Detecting and inhibiting viral translocation promoting agents - specifically mammalian G-protein-coupled chemokine receptors which interact with CD4 to facilitate infection by **human immunodeficiency virus.**
DC B04 D16 P14 S03
IN DENG, H; ELLMEIER, W; LANDAU, N R; LITTMAN, D R; LIU, R
PA (AARO-N) AARON DIAMOND AIDS RES CENT; (UYNY) UNIV NEW YORK STATE
CYC 67
PI WO 9744055 A1 19971127 (199802)* EN 84
RW: AT BE CH DE DK EA ES FI FR GB GH GR IE IT KE LS LU MC MW NL OA PT SD SE SZ UG
W: AL AU BA BB BG BR CA CN CU CZ EE GE GH HU IL IS JP KP KR LC LK LR LT LV MG MK MN MX NO NZ PL RO SG SI SK TR TT UA UZ VN YU
AU 9731434 A 19971209 (199824)
US 5939320 A 19990817 (199939)
ADT WO 9744055 A1 WO 1997-US8926 19970520; AU 9731434 A AU 1997-31434 19970520; US 5939320 A **Provisional US 1996-17157P 19960520, CIP of US 1996-650412 19960520, US 1996-666020 19960619**
FDT AU 9731434 A Based on WO 9744055
PRAI US 1997-858660 19970519; US 1996-17157P 19960520;
US 1996-650412 19960520; US 1996-666020 19960619

=> d his

(FILE 'HOME' ENTERED AT 13:34:37 ON 22 JAN 2006)

FILE 'USPATFULL' ENTERED AT 13:34:46 ON 22 JAN 2006
E ALLAWAY G P/IN
L1 22 S E4
E LITWIN V M/IN
L2 9 S E4
L3 1 S L2 NOT L1
E MADDON P J/IN
L4 47 S E4
L5 33 S L4 NOT (L1 OR L2)
L6 11 S L5 AND (CCR5 OR CC-CKR-5 OR CKR5)
L7 43461 S (HIV OR HUMAN IMMUNODEFICIENCY VIRUS)
L8 1714 S L7 AND (CCR5 OR CC-CKR-5 OR CKR5)
L9 10 S L8 AND AY<1997
L10 10 S L9 NOT L1

FILE 'MEDLINE' ENTERED AT 13:41:48 ON 22 JAN 2006
E ALLAWAY G P/AU
L11 28 S E3-E5
L12 4 S L11 AND (CCR5 OR CC-CKR-5 OR CKR5)
E LITWIN V M/AU
E MADDON P J/AU
L13 43 S E2-E4
L14 33 S L13 NOT L11
L15 3 S L14 AND (CCR5 OR CC-CKR-5 OR CKR5)
L16 160375 S (HIV OR HUMAN IMMUNODEFICIENCY VIRUS)
L17 2456 S L16 AND (CCR5 OR CC-CKR-5 OR CKR5)
L18 36 S L17 AND PY<1997

FILE 'WPIDS' ENTERED AT 13:45:59 ON 22 JAN 2006
E ALLAWAY G P/IN
L19 22 S E3
L20 6 S L19 AND (CCR5 OR CC-CKR-5 OR CKR5)
E LITWIN V M/IN
L21 10 S E3

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                E MADDON P J/IN
L23           47 S E3-E4
L24           33 S L23 NOT L19
L25            7 S L24 AND (CCR5 OR CC-CKR-5 OR CKR5)
L26        21982 S (HIV OR HUMAN IMMUNODEFICIENCY VIRUS)
L27          353 S L26 AND (CCR5 OR CC-CKR-5 OR CKR5)
L28            0 S L27 AND AY=1995
L29           12 S L27 AND AY=1996
L30            9 S L29 NOT L19

=> log off
ALL L# QUERIES AND ANSWER SETS ARE DELETED AT LOGOFF
LOGOFF? (Y)/N/HOLD:y
STN INTERNATIONAL LOGOFF AT 13:50:58 ON 22 JAN 2006
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